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## Table of Contents.





## THE MORTALITY IN AUSTRALIA FROM CANCERS PECULIAR TO THE FEMALE.


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A PREVIOUS PAPER (Lancaster, 1950) has already dealt with the mortality from cancer as a whole. The discussion of the sources of data in Australian official statistics need not be repeated. It is proposed here to analyse the mortality from cancers of the breast and female genital organs.

### Definitions of the Cancers.

The coding manual of the Registrar-General of England and Wales (1940) includes under the rubric "cancer of the breast", the terms "*cancer en cuirasse*", "cancer of the mamma or mammary gland", "cancer of the nipple", "duct cancer" and "Paget's disease of the nipple". The last two items are distinguished by an asterisk, which indicates that the office of the Registrar-General always seeks further information when such a cause of death is given on the death certificate. A later note states that "cancer of the heart" is always to be queried, for it is often found to be miscopied in transcription or badly written by the certifying medical officer in cases in which he actually wrote or intended to write "cancer of the breast".

The cancers of individual genital organs that can be traced in Australia through the years 1908 to 1945 are those of the uterus, ovaries, vagina and vulva. It seems unlikely that much reliance can be placed upon the division in the more recent years of the cancers of the uterus into cancer of the body and cancer of the cervix. In this paper, cancer of the uterus will be treated as a single entity.

Tabulations have been made irregularly in *Demography*, the annual bulletin of the Bureau of Census and Statistics, Canberra, of the cancers of atypical sites, such as the broad ligament, or of rarer cancers, such as chorion carcinoma. In the earlier years there were also a number of cancers of the "abdomen" or cancers of the "pelvis" recorded. It is probable that the "cancer of the abdomen" was often cancer of the ovaries, and "cancer of the pelvis" was cancer either of the ovaries or of the uterus. It is fortunate that the number of these cases was not at any time large, and they have been neglected in general here, but are included in some of the tables to give totals for the cancers of the female genital organs.

### The Age Distribution of the Deaths from Cancer.

It is convenient to expand here the consideration on the incidence of cancer by age, briefly touched on in the previous paper. If attention is fixed on the mortality from cancer in a particular calendar period, such as the years 1931 to 1940, the mortality for either sex may be considered by age. The number of deaths by age is, of course, the information available from the official statistics; but it is clear that comparisons of the death rates at each age are more fundamental, since they are independent of any peculiarities in the age distribution of the population being studied. The effect of the age distribution of a population is exemplified in Table I, in which the number of deaths at ages as observed in an average million in the population of females in Australia has been compared with the number that would be expected to occur in a million females in the life table population. The age distributions of the deaths in actual populations are the ones usually studied, since they are the ones observed; but Table I shows how arbitrary such age distributions are, since they necessarily depend on the age constitution or distribution of the populations observed. Rather we should like to know at what

ages deaths would occur from cancer in any group of persons that might be followed throughout life. The age distribution of the deaths in the life-table population, which has been used as a standard in the previous paper, is the same as that of the deaths in a number of persons followed in an imaginary population throughout life at the rates of mortality holding in 1932 to 1934, and of the cancer death rates for the same years, which have been taken here to be the same as those of 1931 to 1940. In Figure I are shown graphically the cancer death-rates by age and then the expected number of deaths in each five-year age group in the standard life-table population. It will be seen that the cancer death rates increase throughout life, but that the deaths attain a maximum and then decline. This holds, of course, because the expected number of deaths is equal to the rate multiplied by the number of persons in the age group, and the number of persons in each age group steadily declines throughout life in the life-table population, just as it tends to do in any actual population in the absence of drastic migration. This method of displaying the mortality from cancer has been described here at some length, since it is the most convenient method of comparing the age distribution of the cancers of the individual organs. In Figures IIA and IIB the age distributions of the various cancers are displayed. The rates have all been derived from the deaths in Australia for females in the years 1931 to 1940, and so are strictly comparable among themselves. Figures IIA and IIB are drawn to a vertical scale, such that a given height in Figure I represents seven times the number of deaths that the same height does in Figure IIA or IIB.

TABLE I.

*The Deaths at Ages from Cancer in (a) One Million of the Same Average Age Distribution as Obtained in Australia in the Years 1931 to 1940, and (b) in a Life-Table Population.*

| Age Group. (Years.) | Number of Deaths in the Million Taken as Typical of the Female Australian Population Over the Years 1931 to 1940. | Number of Deaths to be Expected in the Life-Table Population Constructed from the Female Life Tables of the 1933 Census. |
|---|---|---|
| 0 to 14 .. .. .. | 5 (0·5)[1] | 4 (0·2)[1] |
| 15 to 24 .. .. .. | 6 (0·6) | 5 (0·3) |
| 25 to 34 .. .. .. | 20 (1·9) | 18 (1·0) |
| 35 to 44 .. .. .. | 83 (7·7) | 78 (4·3) |
| 45 to 54 .. .. .. | 187 (17·4) | 201 (11·1) |
| 55 to 64 .. .. .. | 253 (23·6) | 365 (20·1) |
| 65 to 74 .. .. .. | 301 (28·1) | 535 (29·5) |
| 75 onwards .. .. .. | 217 (20·2) | 607 (33·5) |
| Total .. .. | 1072 (100·0) | 1813 (100·0) |

[1] The percentages of the deaths by age are given in parentheses.

It appears that the cancers of the breast and uterus and ovaries are each of greater importance from a mortality point of view than any other single cancer up to the late forties, when cancer of the stomach and duodenum and cancer of the intestines (excluding the rectum) attain an importance equal to that of the ovaries, and finally at ages over seventy years these two classes of alimentary cancer are each of greater importance than any other. Combining the deaths at all ages, we find that the deaths in the life-table population as measured by the standardized death rates from the cancers of the various organs may be given in order of importance as follows: (i) stomach and duodenum, 351; (ii) breast, 327; (iii) intestines, excluding rectum, 285; (iv) uterus, 235; (v) liver, 102; (vi) ovaries, 72; (vii) rectum, 70; (viii) pancreas, 58; (ix) skin, 57; (x) respiratory tract, 39; the number in each case is the standardized death rate *per annum* per million. Thus, if a group of females was followed throughout their life, it would be expected that of all the various sites of cancer, the stomach would claim the greatest number of victims, being followed closely by the breast, the intestines (excluding the rectum) and the uterus. If the ages of the females dying are considered, greater importance is to be ascribed to cancers of the breast or uterus than to any of the others, for they tend to occur in younger women. The cancers peculiar to the female are obviously a very important part of female cancer mortality.

In Figure I an arithmetic grid has been used. The curve of the mortality rate is such that it is concave upwards, because the rate of increase of the rates with age is continually increasing. This may be compared with Figure II of the previously mentioned article, in which the grid is semi-logarithmic and the total cancer rates rise practically along a straight line with increase of age, indicating that the rates for total cancer mortality rise as a geometric progression with age. With the very steep rise in the later part of the curve in Figure I of the present paper, it is most difficult to make comparisons between different cancers or the same site of cancer at different times. Therefore, as was mentioned in an earlier paragraph, the device of measuring the number of deaths in a standard population can be justified quite apart from the biological considerations by the greater convenience of graphical exposition.

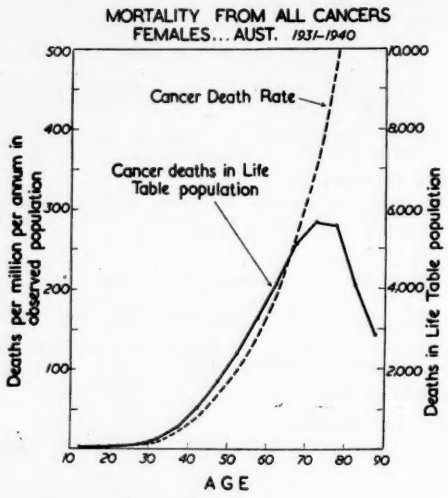


FIGURE I.

(*a*) The mortality from all cancers as a rate per million *per annum* by age, and (*b*) the expected number of deaths *per annum* in a standard million for each five-yearly age group. (Arithmetic grid.)

To illustrate the importance of these cancers, the proportions of the cancer deaths due to the breast and to the female genital organs have been computed and are shown in Table II. In both periods, 1908 to 1930 and 1931 to 1945, between the ages twenty-five and fifty-four years, these two groups of cancers account for some 50% to 60% of all cancer deaths. Cancer of the breast has increased in relative importance between the two periods.

### The Mortality from Cancer of the Breast.

In Table III and in Figure III the death rates from cancer of the breast by age are displayed for five periods between 1908 and 1945. At ages twenty-five to thirty-four years the death rates have remained constant, whereas at every other age the rates have risen. At ages sixty-five to seventy-four years the rates have practically doubled over the period considered. These increases appear to be real increases, for one would not have expected difficulty in the past in the diagnosis of cancer of the breast in subjects who died. Another source of increase may have been the assignment of some of the deaths from cancer of the breast to vaguer terms in the past, such as to cancer of the neck,

axilla, chest, heart, glands of the neck, and so on. But this is not the whole explanation, since in Table IV all these cases have been added in for the two periods 1908 to 1930 and 1931 to 1945, and still there is an increase at every age, in what may be termed death rates from "presumed cancer of the breast". To make comparisons easier, the rates at each age in the later period have been expressed as a percentage of the rates in the earlier period. The relative and absolute increase is greater in the later age groups. In the absence of any sufficient demonstrable change in certification practice, it must be provisionally assumed that there has been a real increase in cancer of the breast. Cumpston (1936) had already noted this increase. In other countries, concern has been expressed about this trend in the breast cancer death rates as measured by the official statistics, and about the low standard of cures as shown by analyses of individual case history records. Thus McKinnon (1949, 1950) has discussed in some detail the official statistics for the Province of Ontario, for Massachusetts and for England and Wales. In both Ontario and Massachusetts, intensive campaigns against cancer have been initiated and carried through with energy. They have been successful in bringing the patient to the doctor at an earlier stage, they have also succeeded in reducing the time between the first visit to the doctor and the commencement of treatment, yet no effective reduction in the cancer rate has ensued. In England, M. Page (1948), in his presidential address to the Section of Surgery of the Royal Society of Medicine, notes that the confident opinions of many surgeons as to the efficacy of surgery are not always founded on adequate follow-up series. It is clear from the discussion that followed that there can be little cause for satisfaction or complacency in the treatment of breast cancers. That the

TABLE II.

*The Percentage of Cancer Deaths in Each Age Group due to Cancer of the Breast or to Cancer of the Female Genital Organs, in Australia.*

| Age Group. (Years.) | Cancer of the Breast. | | Cancer of the Female Genital Organs. | | Total of Cancers Peculiar to the Female Sex. | |
|---|---|---|---|---|---|---|
| | 1908 to 1930. | 1931 to 1945. | 1908 to 1930. | 1931 to 1945. | 1908 to 1930. | 1931 to 1945. |
| 0 to 24 .. | 1·0 | 0·8 | 15·9 | 13·3 | 16·8 | 14·1 |
| 25 to 34 .. | 16·4 | 17·1 | 32·1 | 32·9 | 48·5 | 50·0 |
| 35 to 44 .. | 22·8 | 28·9 | 34·1 | 32·1 | 56·9 | 60·9 |
| 45 to 54 .. | 21·0 | 27·4 | 30·4 | 30·2 | 51·3 | 57·6 |
| 55 to 64 .. | 15·2 | 22·1 | 24·1 | 22·9 | 39·3 | 45·0 |
| 65 to 74 .. | 12·4 | 15·6 | 16·2 | 16·5 | 28·6 | 32·1 |
| 75 and over .. | 12·7 | 14·3 | 11·3 | 11·9 | 24·0 | 26·2 |
| Total .. | 15·9 | 19·7 | 22·6 | 20·7 | 38·6 | 40·4 |

TABLE III.

*Cancer of the Female Breast. Death Rates per Annum per Million at Risk, in Australia.*

| Age Group. (Years.) | 1908 to 1910. | 1911 to 1920. | 1921 to 1930. | 1931 to 1940. | 1941 to 1945. |
|---|---|---|---|---|---|
| 0 to 24 .. | 0 | 0 | 0 | 0 | 0 |
| 25 to 34 .. | 22 | 22 | 23 | 24 | 21 |
| 35 to 44 .. | 155 | 131 | 159 | 170 | 169 |
| 45 to 54 .. | 316 | 337 | 419 | 431 | 442 |
| 55 to 64 .. | 462 | 455 | 574 | 701 | 733 |
| 65 to 74 .. | 510 | 692 | 804 | 915 | 1042 |
| 75 and over .. | 1024 | 1093 | 1247 | 1424 | 1659 |
| All ages .. | 100 | 113 | 156 | 206 | 247 |

TABLE IV.

*The Death Rates from "Presumed Cancer of the Breast"—Cancer of the Breast to Which has been Added Cancer of the Axilla, Chest, Heart, Neck and Glands of the Neck.*

| Age Group. (Years.) | Death Rates from "Presumed Cancer of the Breast" as Deaths per Million *per Annum* for the Periods | | The Death Rates in 1931 to 1945 Expressed as a Percentage of the Rates in 1908 to 1930. |
|---|---|---|---|
| | 1908 to 1930. | 1931 to 1945. | |
| 0 to 24 .. .. | 1 | 0 | — |
| 25 to 34 .. .. | 24 | 23 | — |
| 35 to 44 .. .. | 153 | 170 | 111 |
| 45 to 54 .. .. | 388 | 438 | 113 |
| 55 to 64 .. .. | 544 | 718 | 132 |
| 65 to 74 .. .. | 777 | 973 | 125 |
| 75 and upwards .. | 1270 | 1557 | 123 |
| All Ages .. | 139 | 223 | — |

TABLE V.

*The Mortality Rates from Cancer of the Uterus.*

| Age Group. (Years.) | Deaths per Annum per Million in the Periods | | | | |
|---|---|---|---|---|---|
| | 1908 to 1910. | 1911 to 1920. | 1921 to 1930. | 1931 to 1940. | 1941 to 1945. |
| 0 to 24 .. | 0 | 1 | 1 | 1 | 1 |
| 25 to 34 .. | 33 | 32 | 28 | 28 | 28 |
| 35 to 44 .. | 222 | 181 | 169 | 142 | 122 |
| 45 to 54 .. | 467 | 454 | 409 | 339 | 322 |
| 55 to 64 .. | 697 | 660 | 605 | 528 | 479 |
| 65 to 74 .. | 711 | 667 | 735 | 688 | 739 |
| 75 and over. .. | 695 | 601 | 608 | 857 | 823 |
| All ages .. | 136 | 137 | 148 | 157 | 166 |

TABLE VI.

*Cancer Mortality in Australia, Peculiar to the Female Sex. Deaths from Cancers of the Female Genital Organs, shown as from Individual Organs (Australia).*

| Age Group. (Years.) | Uterus. | | Ovary. | | Vagina. | | Vulva. | | Chorion *et cetera*. | | Abdomen and Pelvis. | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1908 to 1930. | 1931 to 1945. | 1908 to 1930. | 1931 to 1945. | 1908 to 1930. | 1931 to 1945. | 1908 to 1930. | 1931 to 1945. | 1908 to 1930. | 1931 to 1945. | 1908 to 1930. | 1931 to 1945. |
| 0 to 24 .. | 23 | 24 | 57 | 43 | 1 | 2 | 1 | 4 | 2 | 0 | 30 | 8 |
| 25 to 34 .. | 318 | 226 | 76 | 115 | 7 | 3 | 1 | 3 | 2 | 0 | 26 | 7 |
| 35 to 44 .. | 1411 | 961 | 203 | 323 | 16 | 16 | 9 | 12 | 0 | 3 | 102 | 25 |
| 45 to 54 .. | 2429 | 2029 | 385 | 788 | 53 | 24 | 22 | 44 | 2 | 4 | 199 | 40 |
| 55 to 64 .. | 2364 | 2155 | 305 | 750 | 51 | 52 | 62 | 73 | 0 | 2 | 307 | 93 |
| 65 to 74 .. | 1414 | 1850 | 143 | 505 | 41 | 54 | 70 | 140 | 0 | 2 | 252 | 112 |
| 75 and over | 538 | 980 | 51 | 215 | 33 | 43 | 51 | 135 | 2 | 3 | 190 | 96 |
| Unclassified | 7 | — | — | — | — | — | — | — | — | — | — | — |
| All ages .. | 8504 | 8225 | 1220 | 2739 | 202 | 194 | 216 | 411 | 8 | 14 | 1106 | 381 |

problem is of great importance can be seen from the Australian statistics. In the period 1931 to 1940, the standardized death rate from cancer of the breast was 327 per million *per annum*—approximately 2·2% of the total mortality for females from all causes. This indicates that at the rates existing in 1931 to 1940 some 2·2% of all females born in Australia would die of cancer of the breast.

The failure of therapy is thought to lie in the dissemination of the malignant cells before the primary growth can be treated. If, as seems possible, the campaigns for earlier diagnosis fail to reduce the death rate, a more drastic solution may have to be considered. Certainly any breast in which there is the slightest evidence or suspicion of malignant disease or its precursors, should be removed.

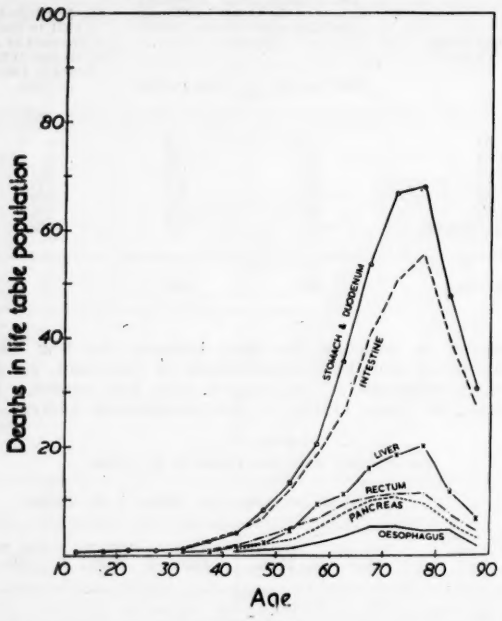


FIGURE IIA.

The deaths by age from certain cancers in the standard population. The standard population is that of the 1933 Australian life tables, and both life-table and cancer rates may be taken to be founded on the experience in Australia of the years 1932 to 1934.

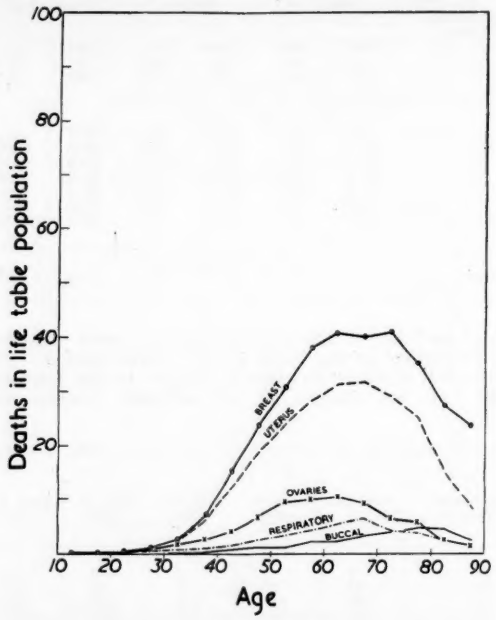


FIGURE IIB.

The deaths by age from certain cancers in the standard population. The standard population is that of the 1933 Australian life tables, and both life-table and cancer rates may be taken to be founded on the experience in Australia of the years 1932 to 1934.

### Cancer of the Uterus.

In Table V are given the death rates for cancer of the uterus over the years 1908 to 1945 in Australia. There has been a definite decline at ages thirty-five to forty-four years and at ages forty-five to fifty-four years, and a less striking decline at ages fifty-five to sixty-four years. The rates in the next age group (sixty-five to seventy-four years) have remained practically stationary, and there appears to have been a rise in the ages above seventy-five years. It is possible that this rise is spurious (see the note in the previous article), and in any case it is not a large increase. The reduction at ages thirty-five to forty-four years is of interest, as it accounts almost entirely for the reduction in the mortality figures for cancer (all forms) over the same periods. This is easily demonstrated by subtracting the uterine cancer rates from the rates for all forms of cancer for each of the periods. When this is done, it is found that the rates from all others forms of cancer have remained practically constant. This finding is consonant with clinical experience, as there are now a number of series of follow-up investigations of cases of uterine cancer treated by surgery, with or without irradiation, in which the five-year or ten-year survival rates show the effectiveness of the therapy.

### Cancer of Other Female Genital Organs.

In Table VI are set out the deaths from the cancers of the female genitalia for the years 1908 to 1930 and 1931 to 1945. Cancer of the uterus is seen to be of greater importance as a cause of death than the others. At ages under

TABLE VII.

*A Comparison of the Total Cancer Mortality and of the Mortality from Cancer of the Ovary, Vagina and Vulva, in Australia for the Years 1908 to 1930 and 1931 to 1945.*

| Age Group. (Years.) | The Death Rate per Million *per Annum* for Cancers. | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | All Forms. | | Ovary. | | Vagina. | | Vulva. | |
| | 1908 to 1930. | 1931 to 1945. | 1908 to 1930. | 1931 to 1945. | 1908 to 1930. | 1931 to 1945. | 1908 to 1930. | 1931 to 1945. |
| 0— | 24 | 28 | 2 | 2 | — | — | — | — |
| 25— | 137 | 133 | 8 | 14 | — | — | — | — |
| 35— | 648 | 588 | 26 | 45 | 2 | 2 | — | — |
| 45— | 1798 | 1590 | 68 | 129 | 9 | 4 | 4 | 7 |
| 55— | 3430 | 3221 | 82 | 177 | 14 | 12 | 17 | 17 |
| 65— | 5941 | 6162 | 72 | 193 | 21 | 21 | 35 | 53 |
| 75+ | 9200 | 10,650 | 62 | 185 | 40 | 37 | 61 | 116 |

twenty-five years, cancer of the ovary is more frequent than cancer of the uterus, but both are relatively unimportant as a cause of death at these ages. Cancers of the vagina and of the vulva are both seen to be concentrated on later age groups than cancer of the uterus. Cancer of the chorion would appear to be a rarity, but is probably often included as a cancer of the uterus without further specification.

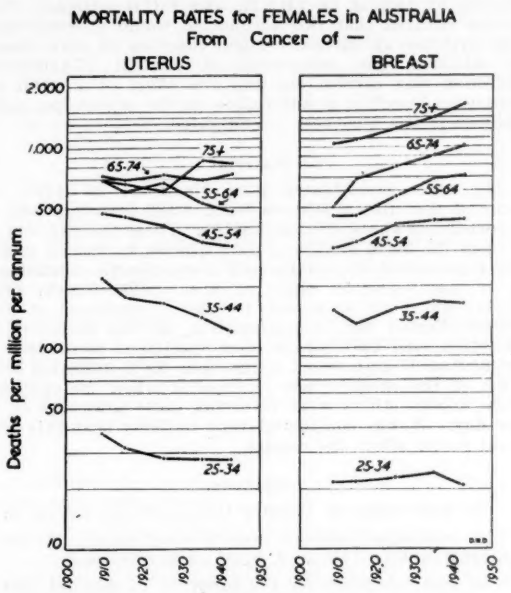


FIGURE III.
The trend of the death rates in Australia from cancer of the breast and from cancer of the uterus. Semi-logarithmic grid.

In Table VII some of these deaths have been expressed as a mortality rate. The most striking feature is the rise in the mortality rates of cancer of the ovary when the two periods are compared whose mid-points lie twenty years apart. This rise has also been noticed in England (Registrar-General, 1949), where the current rates are compared with rates over previous decennia. The trends in the two countries are similar, although the Australian figures are slightly below the English. There does not seem to be any ready explanation of this feature. The age-specific mortality rates from cancer of the vulva and of the vagina in Australia do not show any definite trend, with the single exception of the rise in the death rates from cancer of the vulva at ages over seventy-five years. In the English report the two cancers are combined in a single table, and the figures there are of a magnitude comparable with the Australian figures.

### The Effect of Age, Conjugal Condition and Parity on the Cancer Death Rates.

From the subsidiary tables on cancer mortality in *Demography* dealing with parity and conjugal condition, the observed figures for deaths from cancer in Table VIII have been computed. *Demography* distinguishes between four classes of females: the "never married", the "married with children", the "married without children" and the females of unstated conjugal condition. This last class contains few entries, and in the construction of Table VIII the cancer deaths of women of unstated conjugal condition have been removed wholly from consideration. Corresponding corrections have been made to the grand totals. We then have, for the years 1931 to 1940, the number of deaths by age, by site of cancer and by conjugal condition and parity. The experience of individual calendar years had been pooled by the courtesy of the Commonwealth Statistician (Dr. Roland Wilson). I have further consolidated the data by making the age groups ten-yearly, these groups corresponding to those for which, in earlier tables, the death rates have been computed.

The comparison that comes to mind immediately in treating data of this nature is the death rate in the various classes—that is, the number of deaths in the class divided by the appropriate number of persons at risk. However,

TABLE VIII.
*The Conjugal State of Females Dying of Certain Cancers, in Australia, by Age in the Years 1931 to 1940.*

| Age Group (Years) and Site of Cancer. | Never Married. | Married With Children. | Married Without Children. | Total with Conjugal Condition Stated.[1] |
|---|---|---|---|---|
| *Breast:* | | | | |
| 25 to 34 .. | 22 (29) | 83 (76) | 19 (19) | 124 |
| 35 to 44 .. | 146 (134) | 558 (545) | 90 (115) | 794 |
| 45 to 54 .. | 322 (270) | 1131 (1156) | 206 (233) | 1659 |
| 55 to 64 .. | 402 (311) | 1151 (1263) | 258 (237) | 1811 |
| 65 to 74 .. | 298 (224) | 1034 (1121) | 174 (160) | 1506[2] |
| 75 and over .. | 142 (106) | 713 (786) | 120 (83) | 975 |
| *Uterus:* | | | | |
| 25 to 34 .. | 20 (34) | 97 (89) | 28 (22) | 145 |
| 35 to 44 .. | 69 (112) | 468 (453) | 123 (95) | 660 |
| 45 to 54 .. | 118 (212) | 982 (910) | 206 (183) | 1306 |
| 55 to 64 .. | 198 (234) | 981 (951) | 185 (179) | 1364 |
| 65 to 74 .. | 102 (169) | 824 (843) | 146 (120) | 1132 |
| 75 and over .. | 53 (64) | 481 (473) | 52 (50) | 586 |
| *Cancer of Other Female Genital Organs:* | | | | |
| 25 to 34 .. | 29 (20) | 39 (52) | 17 (13) | 85 |
| 35 to 44 .. | 57 (39) | 133 (160) | 43 (34) | 233 |
| 45 to 54 .. | 139 (90) | 313 (385) | 101 (78) | 553 |
| 55 to 64 .. | 126 (93) | 313 (376) | 101 (71) | 540 |
| 65 to 74 .. | 98 (64) | 275 (319) | 56 (46) | 429 |
| 75 and over .. | 37 (25) | 166 (187) | 29 (20) | 232 |
| *Cancer of All Other Sites:* | | | | |
| 25 to 34 .. | 87 (75) | 191 (194) | 39 (49) | 317 |
| 35 to 44 .. | 197 (183) | 741 (742) | 144 (156) | 1082 |
| 45 to 54 .. | 430 (437) | 1898 (1872) | 357 (377) | 2685 |
| 55 to 64 .. | 718 (806) | 3413 (3268) | 556 (614) | 4687 |
| 65 to 74 .. | 932 (1032) | 5300 (5150) | 686 (736) | 6918 |
| 75 and over .. | 553 (590) | 4460 (4374) | 412 (461) | 5425 |

[1] The cases with conjugal condition or age unstated have been omitted.
[2] The totals of the expected may not always sum up to the observed because of errors introduced by rounding.
[3] The "expected deaths" are shown in parentheses, alongside the observed deaths.

it is doubtful if this would be a fair test, since the presence of a chronic disease such as cancer will hinder the passage of a female from the class of unmarried to one of the married classes, so that the unmarried in the population at large may from this cause come to contain an undue number of cancer patients and so deaths. The never married will be a selected class. A fairer comparison can be made between the proportion of cancers of a certain site in a given age group which occur in unmarried females, and the proportion of all other cancers which appear in this class. So for each age group I have computed the number of cancer deaths of each site that would be expected to occur in each conjugal class if the proportions were the same for each cancer as they are for cancer, all forms. There are thus for each age group a set of observed cancer deaths and a set of expected cancer deaths. Application of $\chi^2$, or indeed inspection of the results, shows that the hypothesis that the incidence of the various sites of cancer is independent of the conjugal condition is not acceptable. However, the tables are not shown in this form, but in one rearranged so that the deaths from cancer of each site are brought together. The expected deaths are shown in parentheses in each case alongside the observed number of deaths. The conclusions to be drawn from Table VIII may be summarized as follows.

*Breast.*

At every age above thirty-five years there is an excess of the breast cancer deaths in the class "never married". In the class "married without children", there is an excess of cancer deaths only in the later years of life—fifty-five

to sixty-four years—and the higher age groups. The excess is large in this class only in the age group seventy-five years and over.

*Uterus.*

At every age there are a deficit of the observed deaths among the "never married" and an excess of deaths among the married, whether they have borne children or not.

*Other Cancers of the Female Genital Organs.*

Other cancers of the female genital organs are largely cancer of the ovary. There is an excess of observed cancers among women who have not borne children whether they are married or not. The class "unmarried" might well be divided up into those who have and those who have not borne children. Such a tabulation would be difficult to obtain.

## Summary.

An attempt has been made to bring out the important features of the mortality of females from cancers of the breast and of the female genital organs, considered at a point of time or over a number of years for evidence of trends.

It has been shown that these cancers form an important part of all female mortality and in particular of female cancer mortality. Cancer of the breast and cancer of the ovaries are both increasing in frequency as a cause of death. At some ages there has been, over the last forty years, a decline in the mortality from cancer of the uterus.

The effect of conjugal condition on frequency has been noted, it being confirmed that cancer of the ovaries is relatively common among those who have not borne children, cancer of the uterus is more common among the married, and cancer of the breast is more common among the "never married".

## Acknowledgements.

Table VIII was constructed from data tabulated by the Bureau of Census and Statistics, Canberra (Dr. Roland Wilson). Mr. D. W. Davies has drawn the diagrams. I have to thank Dr. R. C. Gill and Dr. R. E. Fowler for reading the manuscript. The paper is published with the permission of the Director-General of Health, Dr. A. J. Metcalfe.

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# EVIDENCE OF ADRENAL CORTICAL FUNCTION IN PINK DISEASE.

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PREVIOUS WORK (Cheek and Stace, 1950; Cheek and Hicks, 1950; Cheek, 1951) has revealed evidence for a disturbance of electrolyte and water metabolism in pink disease. The satisfactory clinical results achieved by the therapeutic oral use of salt (approximately eight grammes per day), combined with injections of desoxycorticosterone acetate, support the view that many of the symptoms of pink disease are related to this electrolyte disturbance. The results of investigation and treatment naturally draw attention to the adrenal glands as possibly being of basic importance in the nature of this disease. The rise in the level of the circulating adrenaline in the blood (suggested by other authors) has been shown to be unlikely (Cheek, 1951*b*). In this paper, therefore, investigations are presented in an attempt to clarify further the position of the adrenal cortex in pink disease. In connexion with studies of urinary glucocorticoid excretion in patients suffering from various types of hypertension by two of us (B.S.H. and D.C.H.—Hetzel, 1950), it was realized that this estimation would provide important evidence of adrenal cortical function in pink disease. In addition, the estimation of neutral 17-ketosteroid excretion was carried out and the effect of a small subcutaneous injection of adrenaline on the circulating eosinophile cells has also been investigated.

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## Material.

The cases investigated were diagnosed by D.B.C. and some of the clinical histories and other investigations are reported elsewhere (Cheek, 1951*b*). These patients did not present the florid picture of pink disease as seen in infants, but represented the milder and more chronic condition as it is manifested in older children (*vide* Cheek, 1951). Forty-eight hour or seventy-two hour specimens of urine were collected for the estimation of the glucocorticoid excretion, and twenty-four hour specimens were used for the neutral 17-ketosteroid estimation. As is indicated below, some of the children were untreated when the specimens were taken; others were receiving eight grammes of salt per day. It was considered very unlikely that this treatment would affect the results.

## Methods.

*The Estimation of Urinary Glucocorticoid Excretion.*

The technique followed was a modification of that of Venning *et alii* (1946). A brief outline follows.

The assay depends on the property of adrenal cortical hormone (containing glucocorticoids) of depositing glycogen in the liver in the fasting adrenalectomized mouse. This principle was first used by Reinecke and Kendall (1942) in rats, but with the use of mice and a standard dose of glucose the technique has been made much more sensitive and suitable for the assay of adrenal cortical activity in urine. The extraction of urine was by means of chloroform at pH 1·0. After being washed and dried the extract was stored in the cold room at 4° C. until assayed. The response of the mice was standardized by means of whole aqueous adrenal cortical extract "Eschatin" (Parke, Davis), of which one millilitre was found equivalent in activity to 200 microgrammes of compound "E" (17 hydroxy-11 dehydrocorticosterone). Results were expressed in microgrammes, one microgramme being equivalent to the activity of one microgramme of compound "E". The precision of this standardization was not as great as we should have desired. This will be discussed in another paper (Hetzel and Hine, in preparation).

*The Estimation of Neutral 17-ketosteroid Excretion.*

For estimation of neutral 17-ketosteroid excretion, a modification of the method of Holtorff and Koch (1940) was used without correction for non-specific chromogens.

*The Effect of Adrenaline on the Circulating Eosinophile Cells.*

Thorn *et alii* (1949) showed in man that a fall in the circulating eosinophile cells could be produced by the intramuscular injection of adrenocorticotrophic hormone, and that this phenomenon could also be produced by the administration of the 11 oxysteroid fractions of the adrenal cortex (Hills, Forsham and Finch, 1948). It was known that the same effect could be produced with adrenaline, and this was shown by Recant *et alii* (1948, 1950) to be dependent on pituitary-adrenocortical integrity. This test depended on the degree of fall of the circulating eosinophile cells for hours after a subcutaneous injection of 0·3 milligramme of adrenaline. These authors found that a fall of 50% or more excluded both adrenocortical and pituitary-

TABLE I.

| Pink Disease Child. | Age. (Years.) | Sex. | Mean Mouse Glycogen. (Milligrammes per 100 Grammes.) | Microgrammes of "E" in Twenty-four Hours.[2] | Normal Child of Same Age and Sex. | Mean Mouse Glycogen. (Milligrammes per 100 Grammes.) | Microgrammes of "E" in Twenty-four Hours.[2] |
|---|---|---|---|---|---|---|---|
| M.W.[1] .. .. .. | 2½ | F. | 18·1 | 19 | L.G. | 45·1 | 71 |
| J.K.[1] (i) .. .. .. | 3 | F. | 36·5 | 47 | B.G. | 35·5 | 44 |
| J.K.[1] (ii) .. .. .. | | | 30·1 | 34 | | | |
| E.O. .. .. .. | 2¾ | M. | 30·3 | 34 | R.I. | 28·3 | 31 |
| G.T.[1] .. .. .. | 2½ | F. | 44·9 | 60 | J.C. | 53·1 | 105 |
| K.R. (i) .. .. .. | 2½ | M. | 48·1 | 82 | B.A. | 30·6 | 35 |
| K.R.[1] (ii) .. .. .. | | | 42·2 | 62 | | | |
| M.M.[1] .. .. .. | 2 | F. | 38·1 | 50 | F.J. | 53·5 | 107 |
| M.M. (*a*) (i) .. .. | 3 | F. | 46·7 | 77 | B.C. | 52·9 | 104 |
| M.M.[1] (*a*) (ii) .. .. | | | 21·7 | 23 | | | |
| J.C. .. .. .. | 4 | F. | 38·5 | 51 | J.G. | 21·2 | 22 |
| T.J. .. .. .. | 5 | M. | 44·8 | 70 | D.E. | 59·8 | 145 |
| J.W. .. .. .. | 7 | F. | 28·5 | 32 | B.H. | 36·6 | 47 |

[1] Under treatment with salt, eight grammes per day.

[2] Mean of six mice, each injected with equivalent of eight hours' urine expressed as liver glycogen per 100 grammes mouse weight. Mean deposition, pink disease: 36·04 ± 2·68 (equivalent to 45 microgrammes per day); mean deposition, normal: 41·68 ± 4·11 (equivalent to 60 microgrammes per day); $t = 1·20$—not significant. (The figures 2·68 and 4·11 represent the standard deviation of the mean.)

[3] This column expresses the liver glycogen content in terms of a unit based on the deposition given by one microgramme of compound "E" (17 hydroxy-11 dehydrocorticosterone).

adrenocorticotrophic hormone deficiency. During this investigation in children only 0·2 milligramme of adrenaline has been used.

Direct examination of eosinophile cells, with a Levy counting chamber and eosin-acetone diluting and colouring fluid, was undertaken.

## Results.

The results are shown in Tables I, II and III. We shall discuss each table in turn.

### *The Glucocorticoid Excretion in Urine.*

It will be seen that estimations were made in ten cases of pink disease (three of them twice) and on ten normal children of the same age and sex.

Statistical analysis, carried out by computing the means of the mouse liver glycogen and testing the significance of the difference by the *t* test, showed that though the mean depositions in the pink disease group (36·04 milligrammes per 100 grammes) were less than the normal (41·68 milligrammes per 100 grammes), the difference was not significant. The corresponding figures for microgrammes "E" were 45 microgrammes per day and 60 microgrammes per day respectively.

Venning and Kazmin (1946) found values of 35 to 40 microgrammes per day in two male children between the ages of two and a half and three years, while from the age of five and a half years adult male values were found—40 to 85 microgrammes, average 60 microgrammes. Most of these subjects were females, and it will be seen that adult values (25 to 65 microgrammes, average 40 microgrammes per day) were also obtained.

The differences in the microgramme equivalents from those of Venning and Kasmin (1946) are largely due to the difference in the slope of the regression lines used. However, all the normal children and some of those suffering from pink disease were ambulant. Venning and Kazmin (1946) noted that patients in bed appeared to have a more even output of glucocorticoids than did active normal subjects, probably owing to the fact that the adrenal cortex responds promptly to the everyday stresses of exercise and emotional excitement. This view is supported by the higher standard deviation of the normal group—though the difference is not statistically significant. An occasional high value was probably due to variation within the assay itself—that is, in the response of the mice. This will be discussed more fully in another paper (Hetzel and Hine, in preparation).

### *The 17-Ketosteroid Excretion.*

As there is considerable controversy (Mason and Engstrom, 1950) about the values for children, a series of estimates on normal children of approximately the same age and sex were made at the same time as on the subjects of pink disease.

Statistical analysis, carried out by computing the means and testing the significance of the difference by *t* test, showed a significantly raised excretion of 17-ketosteroid in pink disease. The figures obtained for normal children confirm the few given by Venning and Kazmin (1946), who also used a modification of the method of Holtorff and Koch (1940), but with a correction factor for non-steroidal chromogens. There is probably no great sex difference up to the age of twelve years (Mason and Engstrom, 1950).

The interpretation of this finding is difficult because of the doubt about just what the 17-ketosteroid estimation represents—androgens or metabolites or both, or precursors of other steroids produced by the adrenal cortex.

TABLE II.

| Pink Disease Child. | Age. (Years.) | Sex. | 24-Hour Neutral 17-Ketosteroid Excretion. (Milligrammes.)[2] | Normal Child. | Age. (Years.) | Sex. | 24-Hour Neutral 17-Ketosteroid Excretion. (Milligrammes).[2] |
|---|---|---|---|---|---|---|---|
| M.W.[1] .. .. .. | 2½ | F. | 3·1 | G.T. | 3 | F. | 2·2 |
| J.K.[1] .. .. .. | 3 | F. | 3·2 | J.F. | 3 | F. | 1·2 |
| E.O. .. .. .. | 2¾ | M. | 2·1 | Y.B. | 3 | M. | 0·9 |
| B.M. .. .. .. | 3 | F. | 2·2 | V.U. | 3 | F. | 1·5 |
| G.T. .. .. .. | 2½ | F. | 2·3 | K.W. | 3 | M. | 0·9 |
| J.P. .. .. .. | 3 | F. | 2·8 | K.P. | 3 | F. | 1·2 |
| K.R. .. .. .. | 2½ | M. | 2·3 | J.N. | 3 | M. | 1·5 |
| A.D. .. .. .. | 5½ | F. | 4·8 | K.C. | 5 | F. | 2·1 |
| G.C.[1] .. .. .. | 2½ | F. | 3·8 | B.H. | 3 | F. | 1·3 |
| M.M. .. .. .. | 3 | F. | 2·0 | J.D. | 4 | F. | 1·4 |
| T.J.[1] .. .. .. | 5 | M. | 3·8 | J.E. | 5 | M. | 1·6 |
| J.W. .. .. .. | 7 | F. | 4·2 | D.J. | 7 | F. | 5·3 |

[1] Under treatment with salt, eight grammes per day.

[2] Pink disease mean = 3·05 ± 0·2676; normal mean = 1·76 ± 0·3421; $t = 2·974$ with 22 degrees of freedom—that is, $P$ is less than 0·01. (The figures 0·2676 and 0·3421 represent the standard deviation of the mean.)

*The Effect of Adrenaline on the Circulating Eosinophile Cells.*

Adrenaline, by stimulating the production of adrenocorticotrophic hormone from the pituitary, and therefore the production of glucocorticoids from the adrenal cortex, has produced a demonstrable effect on the circulating eosinophile cells (> 50% in all except one case). In conditions of adrenal insufficiency such as Addison's disease this effect is not obtained with such small doses.

## Discussion.

Altered states of adrenocortical secretion in infancy and childhood are becoming increasingly recognized, and reports of cases are quite frequent. These cases include the following: (i) hypofunction of the adrenal cortex relating only to "the sodium and water hormone"

TABLE III.

| Pink Disease Child. | Age in Years. | Blood Eosinophile Cell Count per Cubic Millimetre. | Blood Eosinophile Cell Count Four Hours After 0·2 Milligramme Adrenaline Injected Subcutaneously. | Percentage Fall. |
|---|---|---|---|---|
| M.W. .. .. | 2½ | 670 | 240 | 64 |
| J.K. .. .. | 3 | 600 | 330 | 45 |
| E.O. .. .. | 2¾ | 350 | 150 | 57 |
| B.M. .. .. | 3 | 900 | 450 | 50 |
| T.J. .. .. | 5 | 670 | 240 | 64 |
| G.T. .. .. | 2½ | 500 | 220 | 56 |
| J.P. .. .. | 3 | 380 | 150 | 60 |
| K.R. .. .. | 2½ | 350 | 120 | 66 |
| A.D. .. .. | 5½ | 550 | 150 | 73 |
| G.C. .. .. | 2½ | 90 | 65 | 28 |
| J.C. .. .. | 4 | 670 | 330 | 51 |
| M.M. .. .. | 3 | 250 | 120 | 52 |
| J.W. .. .. | 7 | 380 | 90 | 76 |

(Jaudon, 1946-1948; Deamer and Silver, 1950); (ii) hyperfunction of the adrenal cortex with excessive production of the androgen hormone; this produces pseudohermaphroditism in girls and sexual precocity in boys (Melicow and Cahill, 1950); (iii) excess production of the androgenic hormone associated with deficiency of the "sodium and water hormone" (for review *vide* Cheek, 1951*a*).

In some reported cases a disturbance of the "sodium and water hormone" is detectable only during periods of "stress" (Geppert *et alii*, 1950; Deamer and Silver, 1950). Generally no alteration in carbohydrate metabolism (as judged by blood sugar levels) is found in cases of *macrogenitosomia præcox* with adrenal insufficiency (Darrow, 1944; Barnett, 1949). The glucocorticoid fraction has therefore been thought to be little disturbed.

Deamer and Silver (1950) recently presented seven cases of altered adrenocortical secretion in childhood with clinical, biochemical and pathological evidence. In five cases an insufficiency of the corticosteroids regulating sodium and water metabolism was demonstrated and blood sugar levels were found to be normal. Concomitant eosinophilia was detected, and three of the five subjects showed sexual and somatic changes.

Table I indicates that glucocorticoid production is normal in pink disease. Such an interpretation obviously is dependent on the precision of the technique used; admittedly the method cannot be regarded as a very precise means of estimating adrenal cortical function, but it is the best technique available as yet. Bornstein and Trewhella (1951) have reported the results of estimations of urinary formaldehydogenic steroids in nine cases of pink disease. They found no difference from normal. As this estimation roughly parallels glucocorticoid production, this result confirms our findings. The findings in Table III provide support for normal glucocorticoid secretion.

The values obtained for the neutral 17-ketosteroid excretion reveal that there is a significant increase in pink disease. This is of interest, in that in conditions of "wasting" and in states of poor nutrition a diminished 17-ketosteroid excretion is the usual finding (Landau *et alii*, 1948). Glanzmann (1937) has noticed the appearance of virilism in some of his cases of infantile acrodynia and presents illustrations of this. Such observations have not been the experience of one of us (D.B.C.). On the other hand, Deamer and Silver (1950) reported three cases in which deficiency of the "salt and water hormone" was accompanied by excessive secretion of neutral 17-ketosteroids. Two of these patients were females in whim pseudohermaphroditism was produced, and the other was a male with sexual precocity. A satisfactory eosinophile cell depression following ACTH was not obtained in the one case that was investigated. Dissociation between excessive 17-ketosteroid excretion and normal glucocorticoid output is characteristic of the adreno-genital syndrome (Venning and Browne, 1947).

Hence one cannot exclude the presence of a fractional hypofunction of the adrenal cortex relating to the sodium and water hormones, although the finding of normal potassium levels is against such an hypothesis (Cheek and Stace, 1950).

On the other hand, sodium depletion in pink disease may primarily result from extraadrenal causes, and this gland may well be embarrassed as a result of these factors. Under such circumstances, salt and adrenal cortical extract would naturally restore plasma osmotic base and correct hæmoconcentration.

## Conclusions and Summary.

Investigation of ten children with pink disease has revealed normal urinary glucocorticoid excretion. The neutral 17-ketosteroid excretion is significantly raised. Response of the circulating eosinophile cells to 0·2 milligramme of adrenaline injected subcutaneously has been normal. It is concluded that in pink disease there is no simple hypofunction of the adrenal cortex. In the light of relevant literature, the possibility of a fractional disturbance of the "sodium and water hormone" should be considered, and also the possible activity of extraadrenal agencies on sodium metabolism.

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## DIFFUSE DISSEMINATED PLATELET THROMBOSIS (THROMBOTIC THROMBOCYTOPENIC PURPURA), WITH A REPORT OF TWO CASES.

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THE recent differentiation by American observers of another clear-cut entity among the conditions characterized by purpura may throw light on the classification and understanding of the nature of these diseases (*Annals of Internal Medicine*, 1950).

This clinical and pathological entity, now known as thrombotic thrombocytopenic purpura, or diffuse disseminated platelet thrombosis, was first described by Moschowitz (1925), who found multiple hyaline thrombi widely disseminated in the organs of a girl, aged sixteen years, who died after a fulminating illness of two weeks' duration, characterized by fever, anæmia and terminal neurological signs, including hemiparesis and coma. In recent years more cases have been reported, but to date there have been only 25 cases in the American literature (*Annals of Internal Medicine*, 1950), and I have been unable to find any reports in British journals.

The main features of the disease have now been established. It appears most commonly in young adult female subjects, but has been observed in both sexes and at ages of from nine to fifty years. The disease generally commences abruptly (*Annals of Internal Medicine*, 1950), often after a preliminary period of vague malaise (Gore, 1950; Baer *et alii*, 1936); occasionally it has developed in a patient with a chronic illness. The most common symptoms are malaise, pyrexia, weakness or even transient paresis (Muirhead *et alii*, 1948), joint pains and a purpuric rash. A severe disorder of the peripheral blood is constantly present, the most obvious feature of which is an anæmia which is rapidly progressive and becomes very severe.

The joint symptoms may not be obvious, or they may be severe enough to prevent movement of the affected part, which becomes swollen and tender. Except in cases in which previous arthritis is present, they are of short duration. The purpura generally takes the form of a rash on the lower part of the legs and the abdomen. Ecchymoses may be present. Abdominal pain, nausea or diarrhœa is frequently complained of, and hæmatemesis or melæna may be present, whilst microscopic hæmaturia is constant.

A peculiar clinical feature is the appearance of bizarre and very varied neurological disturbances (Adams *et alii*, 1948). Pareses and paralyses are common, and may include weakness or spasticity in one or more extremities, cranial nerve palsies and hemiplegia. Convulsions, coma, delirium, dizziness and headache have also been described. These symptoms wax rapidly in severity and also wane in an unpredictable fashion. The cerebro-spinal fluid has been normal in those cases in which it was examined, save for one case in which there had been bleeding into the meninges.

In some cases in which the disorder of the blood picture was obvious from the first and constituted the most notable feature of the disease, the hæmatological investigations have been very fully reported (Fitzgerald *et alii*, 1947; Muirhead *et alii*, 1948), and at the present time it is the disorder of the peripheral blood which is thought to be the most constant and pronounced feature of the disease. The two most prominent features are thrombocytopenia and anæmia. The anæmia is rapidly progressive and is hæmolytic in nature, the red cell count in most cases falling to two million per cubic millimetre or less. It is normocytic and normochromic. Spherocytosis has been reported in two cases (*Annals of Internal Medicine*, 1950), and it was also noted in one of the cases reported here. The reticulocytes are increased in number and normoblasts may even be present in late cases. The patient may be slightly jaundiced.

There is said to be a progressive diminution in the number of platelets, which are virtually absent in the advanced stages. However, this has not been so in at least one case (Muirhead *et alii*, 1948). Associated with this diminution in the number of platelets there is a prolonged bleeding time. A further feature of the disease is the presence in some cases of diffuse proliferative glomerulitis.

The course of the disease is rapidly progressive, the duration varying from one week to two months. Milder types have not been recognized.

The essential histological feature is the presence of hyaline thrombi in the terminal arterioles and capillaries and to a much less extent in the venules. They are widely scattered throughout the body, being most pronounced in the myocardium, renal cortex, adrenals and pancreas.

The primary site of the disease lies in the endothelium of the small vessels, on which an accumulation of platelets takes place. This probably accounts for the thrombocytopenia, the platelets being taken from the blood more quickly than they can be replaced.

The ætiology of the disease is quite unknown, although hypersensitivity has been suggested. All treatment has been of no avail.

### Reports of Cases.

CASE I.—The patient, F.K., was an adult male, aged twenty-two years, single, and a carpenter by trade. He was admitted to hospital on November 21, 1950, because of stiffness and pain in the limbs, which had been diagnosed elsewhere as possibly being due to early tetanus.

The history had begun some three or four weeks previously, when the patient had developed a pain in the back which lasted for several hours and then disappeared. It was unaccompanied by other symptoms. Fourteen days prior to his admission to hospital the patient sustained an abrasion of his right shin, which became infected, causing him to seek medical advice five days later. At this time he was suffering from malaise and had inguinal adenitis. He was treated by daily injections of procaine penicillin, to a total of 2,400,000 units. Three days prior to his admission to hospital he developed backache, aching in the hands and calves of the legs, and stiffness in all his limbs. The next day he developed a fine purpuric rash about his feet and lower limbs. Further questioning revealed that he had had penicillin previously without ill effect. There were no urinary symptoms, and there was no history of allergic phenomena.

Physical examination revealed that the patient was a very tall, muscular man of healthy appearance. There was swelling of both wrists and of the dorsum of the hands. His ankles also were swollen. The ankles were painful to the slightest movement, and the muscles of his limbs were tender, but not in spasm. There was slight generalized abdominal tenderness. The systolic blood pressure was 135 millimetres of mercury and the diastolic 90 millimetres. The temperature was 98·9° F.; the pulse rate was 80 beats per minute; the respiration rate was 20 per minute. Laboratory examination of his urine showed pronounced albuminuria and many red blood cells.

On the day of admission to hospital, the patient developed severe pain and tenderness in the region of his right kidney, which continued up to the time of his death. The pain and swelling of his joints gradually subsided. The rash, which also appeared after his admission to hospital as small, pin-head areas of infiltration on his elbows and face, gradually disappeared. He developed a low-grade pyrexia, which was thought to respond to the exhibition of penicillin, 40,000 units given intramuscularly every four hours.

FIGURE I.

Case I. Pancreas showing earliest change. Vessel with hyaline material beneath the endothelium. (× 717.)

Ten days after admission to hospital, he still had pyrexia and renal pain, with red cells and albumin in his urine. On that day he had an attack of diarrhœa with blood in the stools, from which no pathogenic organism was isolated. The hæmoglobin value was 15·4 grammes per 100 millilitres. A blood count was made on the fifteenth day after admission to hospital, as the patient showed signs of increasing pallor. The result of the count in full was as follows: leucocytes, 29,800 per cubic millimetre, made up of neutrophile cells 77%, lymphocytes 20% and monocytes 3%; erythrocytes, 3,220,000 per cubic millimetre; hæmoglobin value, 9·2 grammes per 100 millilitres; colour index, 1·0; reticulocytes, 9·5%. The erythrocyte fragility was normal, as was the bleeding time. Numerous spherocytes were seen, together with much anisocytosis and poikilocytosis. No deficiency was noted in the number of platelets when the blood was examined, but unfortunately a platelet count was not made, for at that time there was no evidence of a tendency to bleeding.

Two days after this count was made, the patient developed sudden and complete suppression of urine. Three days later, on the twentieth day after admission to hospital, he was beginning to show evidence of renal failure. He had been vomiting for twelve hours, but was still perfectly clear mentally. At that time he suddenly complained that he was unable to move his legs. Within twenty minutes all his limbs were paralysed, and his respiration was slow and gasping. Within thirty minutes he was dead.

Examined during the last stages, he was found to have flaccid paralysis of all his limbs. The deep reflexes were abolished. He appeared to have slight neck rigidity, but Kernig's sign was absent. The blood pressure was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic. The blood urea content at the time of death on December 10 was 200 milligrammes per 100 millilitres of blood.

For treatment "Benadryl", two capsules six-hourly, was given for three days after admission, when he was thought to be suffering from some allergic condition due to penicillin. A mixture containing sodium salicylate 15 grains was given four-hourly for the same period. Penicillin was administered, as mentioned above, until the day of onset of the anuria. Otherwise the treatment was sedative and dietary.

*Post-Mortem Examination.*

Autopsy (of which relevant features only are reported) revealed swollen, congested kidneys dotted with small, punctate hæmorrhages. There was much blood in the renal pelvis. No other abnormality was detected macroscopically, but the microscopic changes were most interesting. In only three organs—the pancreas, the mesenteric lymph glands and, most of all, the kidneys—were the changes pronounced. The primary site of the disease appeared to lie in the small arterioles and capillaries, and all stages were present, from the very earliest up to old, healed lesions. The earliest changes were present in greatest numbers in the pancreas. Here the capillaries were frequently affected. The primary change appeared to be a circular accumulation of hyaline material beneath the endothelium (Figure I). Inside this there was an irregular accumulation of very similar material, probably platelets in later stages. The next stage was one of complete occlusion of the lumina of the vessels by what appeared to be platelet thrombi. In many places there was no surrounding cellular reaction, nor even, as reported (Fitzgerald *et alii*, 1947; Muirhead *et alii*, 1948), any endothelial proliferation (Figure III).

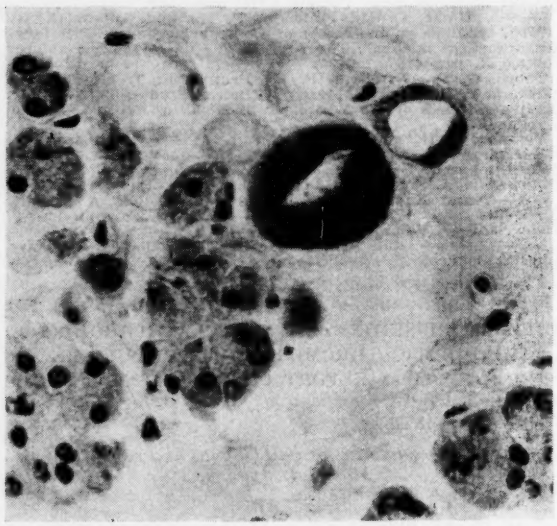

FIGURE II.

Case I. Pancreas showing deposit of hyaline material (platelets) on the affected vessel wall. (× 297.)

In other areas, however, particularly in the kidney and the lymph nodes, lesions typical of *periarteritis nodosa* were to be seen (Figure IV), with fibrinoid degeneration of the arteriole wall and pronounced perivascular cellular infiltration with polymorphonuclear and mononuclear cells. Also present were all degrees of intermediate gradation, from very slight cellular infiltration to pronounced infiltration around a thrombus (Figure V). Indeed, most of the abnormal vessels were in this category.

In the kidney and pancreas there were small areas of infarction, but the other organs had for the most part escaped severe damage. The kidney, in addition, showed a proliferative glomerulitis. The brain and cord and the liver were normal. Changes in the lungs were minimal.

CASE II.—The patient, F.C., was an adult male, aged forty-nine years and married. His occupation was given as business manager. He was admitted to hospital on December 12, 1950. Three weeks prior to his admission he had noticed a sore throat and swelling in the neck, for which he was given penicillin intramuscularly and a course of forty tablets of a sulphonamide drug. The throat improved, but the patient felt generally unwell. Eleven days later he noticed that his urine was pink. It diminished in quantity, but at the same time he had increased frequency of micturition. There were no further symptoms. Sulphonamide administered once previously years before had caused "conjunctivitis".

On examination he was seen to be a rather stout man of the stated age, in no great distress. The systolic blood pressure was 190 millimetres of mercury and the diastolic 110 millimetres. There were blood and albumin in his urine. Pulse, temperature and respiration were normal. A diagnosis of acute glomerulonephritis was made on the clinical findings.

Examination of a blood sample taken on the day of admission showed a total leucocyte count of 11,900 per cubic millimetre, made up of neutrophile cells 69%, lymphocytes 29% and monocytes 2%, a hæmoglobin value of 11·4 grammes per 100 millilitres, a blood sedimentation rate of 50 millimetres in one hour, and a normal blood picture.

The patient's condition was stationary until four days after admission to hospital, when he developed severe pain running from the left loin to the groin, with much tenderness in this area. Morphine was required to give relief. The patient passed only 14 ounces of urine on that day, but the output was increased over the next few days. By the tenth

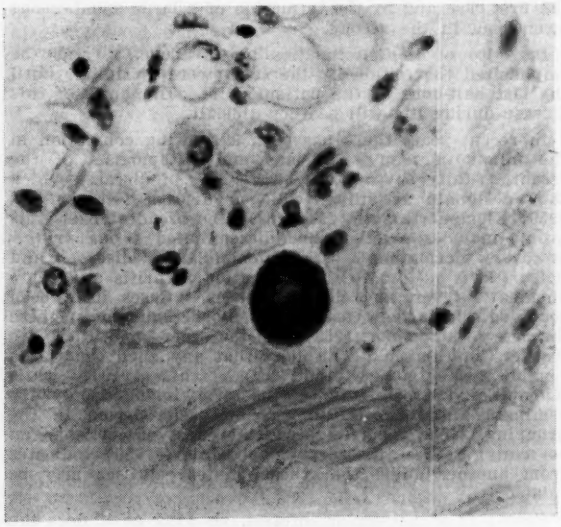

FIGURE III.

Case I. Pancreas showing complete occlusion by thrombus; no surrounding reaction. (× 297.)

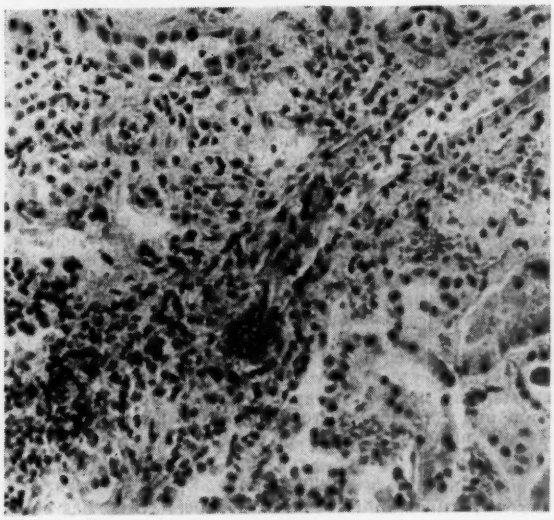

FIGURE IV.

Case I. Arteriole in kidney showing appearance strongly suggestive of *palyarteritis nodosa*. (× 297.)

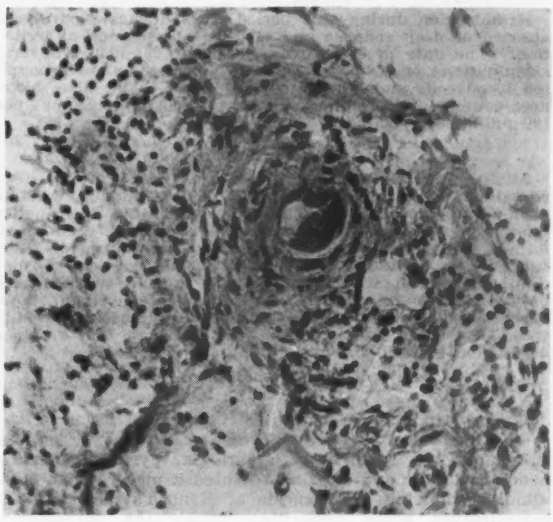

FIGURE V.

Case I. Section of retroperitoneal tissue showing vessel partly occluded by thrombus with mild surrounding inflammatory exudate. (× 297.)

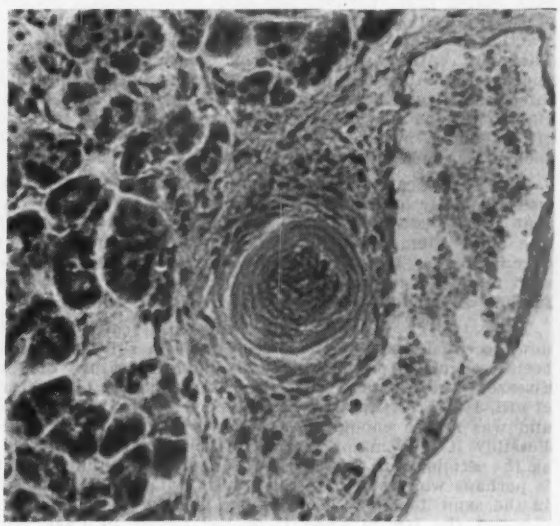

FIGURE VI.

Case II. Section of pancreas showing typical degeneration of vessel wall with occlusion by hyaline thrombus. (× 297.)

day after his admission to hospital the urinary output had fallen once more. The patient was vomiting, hiccuping and obviously developing renal failure. Two days later, while still mentally quite clear, he complained of inability to move his legs. The paralysis spread rapidly over the whole body, death occurring within half an hour.

Examination during this period showed flaccid paralysis, absence of deep reflexes and no signs of meningeal irritation. The date of death was December 25, 1950. Several examinations of his urine during the illness showed many red blood cells and leucocytes, and some casts. The blood urea content at the time of death was 230 milligrammes per 100 millilitres.

*Post-Mortem Examination.*

Autopsy (of which relevant features only are reported) revealed a large hæmatoma around the left kidney, with no obvious bleeding point. The most noticeable abnormality microscopically was pronounced arteriosclerosis, seen in all organs. The kidneys showed signs of late acute or early subacute glomerulonephritis with typical crescent formation by proliferated cells of Bowman's capsule, superimposed on the changes of mild nephrosclerosis. Once again, however, in the small arterioles and in the capillaries, especially those of the pancreas and the kidney, there was evidence of hyaline platelet thrombosis (Figure VI).

The brain and spinal cord showed no abnormality.

**Comment.**

The diagnosis in Case I presents unusual difficulties. Clinically, the features of arthralgia, pyrexia, purpura, abdominal colic and anuria presented a picture that fitted almost completely a diagnosis of Henoch's purpura with complicating acute nephritis. When the pathological findings were known it was easy to fit the case into the category of disseminated platelet thrombosis. This explained some previously obscure features—in particular the sudden, presumably hæmolytic, anæmia with spherocytosis and the peculiar form of death, with its bizarre neurological signs.

However, the very marked perivascular reaction and the typical polyarteritic features in certain areas are changes not previously reported in this disease. Indeed, it has been stated that "perhaps the most characteristic feature of the lesions is the virtual absence of any inflammatory reaction in the walls of the vessels and adjacent tissues. They are entirely different from those of periarteritis nodosa, disseminated lupus erythematosus and of the usual infectious diseases" (*Annals of Internal Medicine*, 1950). Having regard to the findings in this case, and in view of the small number of cases reported, it is felt that such a statement may be premature.

Case II, apart from the exactly similar mode of death, was clinically quite different from Case I, and at the time of the patient's death was thought to be a case of late acute glomerulo-nephritis. The case is not so well documented as Case I, but the histopathological changes seen in the tissue sections leave no doubt that this was a further case of diffuse disseminated platelet thrombosis.

These two cases thus emphasize certain features of the now accepted form of the disease. Glomerulitis has not been recognized previously as a common feature of the disease, although its presence has been reported (Muirhead *et alii*, 1948). It was very pronounced in both these cases and was severe enough to produce renal failure in both. Possibly it is a major feature of the syndrome. Again, as the ætiology of this disease is completely unknown, it is perhaps worthy of note that these two cases both began in the same fashion. There was a history of acute infection—of the leg in Case I, of the throat in Case II—followed by treatment with drugs—procaine penicillin in the one, penicillin and sulphonamide in the other. Although this does no more than suggest an allergic basis for the cause of the disease, it is an aspect which should be followed up.

Of particular interest to me, since it was this which led to the recognition of the disease, was the extreme similarity of the terminal neurological signs, for which no adequate explanation can be given. Hitherto these signs have been characterized by their great variation. These are, so far as I can learn, the first two cases which have shown exactly the same terminal picture.

The bleeding tendency, so notable a symptom in previous reports, was in reality a minor feature in these two cases, being manifested by a purpuric rash of short duration in the first case and by the formation of a large spontaneous hæmatoma in the second.

In spite of the similarities noted above, it must be emphasized that clinically the cases were dissimilar until the last half-hour of the patient's life. Diagnosis of this disease during life will be very difficult.

In recent years there has been a growing conviction in the minds of many clinicians and pathologists of the essential unity as to cause of a series of diseases which they designate as diffuse collagen disease (Kampmeier, 1950; Klempner *et alii*, 1942). On the broadest basis this group includes scleroderma, disseminated *lupus erythematosus*, dermatomyositis, Libman-Sacks disease and *polyarteritis nodosa*, with rheumatoid arthritis and acute rheumatic fever, as related diseases. Cases have been recorded which show features of more than one of these diseases (Kampmeier, 1950). On the other hand, there are workers in this field who deny that any such concept is tenable (Montgomery and McCreight, 1919), while Klemperer, the originator of the term, included only disseminated *lupus erythematosus* and scleroderma in the grouping. The reported beneficial effect of cortisone on so many of these diseases (Mayo Clinic, 1950) indicates from another angle that these clinical entities may be related.

Case I is of particular interest, showing as it does the presence of both platelet thrombosis and perivascular infiltration in the one patient, thus creating a link between this rare disease and *polyarteritis nodosa*. It is suggested, in view of the findings in this case, that diffuse disseminated platelet thrombosis may also be related to the group of collagen diseases, forming, possibly, one end of the scale which has scleroderma at the other, the former showing intravascular changes almost exclusively and the latter showing the changes mainly in the connective tissue of the body.

Another line of reasoning, purely speculative, which is of interest arises from the clinical diagnosis of Henoch-Schönlein purpura which was made in the first case. The patient presented a typical picture of the disease in its more severe form, with complicating acute nephritis. There are a number of ill-defined clinical entities classed under the generic name of anaphylactoid purpura, for which at the present time no uniform pathological basis has been determined. These diseases include, in ascending order of severity, (i) *purpura simplex*, (ii) Henoch's purpura, and (iii) Schönlein's disease. It is interesting to note that in his excellent review of the subject Gairdner (1948) includes Henoch-Schönlein's purpura, acute nephritis, rheumatic fever and *polyarteritis nodosa* in a family of diseases. The histological changes described are not identical with those seen in the cases reported above, but this disease is often mild. Might it not be that milder, non-fatal forms of diffuse disseminated platelet thrombosis have been included in this category? For with a less severe process the platelets may not be so rapidly washed from the bloodstream and the blood picture may appear normal. The answer is not determined.

**Summary.**

Recently a new clinical and pathological entity has been differentiated by American observers. Its principal clinical features are a rapid onset with malaise and pyrexia, joint pains and a purpuric rash. Hæmolytic anæmia and thrombocytopenia develop, and the disease follows a rapid course, ending with obscure neurological signs followed by death. The essential feature found at autopsy is the presence of platelet thrombi widely disseminated throughout the small arterioles, capillaries and venules of the body. To this entity the name thrombotic thrombocytopenic purpura, or diffuse disseminated platelet thrombosis, has been given.

Two cases of this disease are described, both ending in renal failure, paralysis of rapid onset, and death. Autopsy revealed diffuse disseminated platelet thrombosis in both cases, while one case also presented histological features showing an affinity with *polyarteritis nodosa*. The presence of the renal damage is emphasized on this evidence. It is also suggested that this new disease may be related to diseases of the diffuse collagen group, and speculation is made as to its possible relation to the group of anaphylactoid purpuras.

**Acknowledgements.**

I am indebted to Dr. C. J. M. Walters, General Superintendent of the Prince Henry Hospital, for permission to publish this article; to Dr. J. P. O'Brien, late pathologist of this hospital, who first drew attention to the possibility of this disease in Case I; and to Dr. J. Isbister for his advice and encouragement in the preparation of the article.

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## ŒSOPHAGECTOMY FOR CARCINOMA: SOME POINTS IN THE MANAGEMENT.

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FOLLOWING the work of Adams and Phemister (1938) transthoracic œsophagectomy with restoration of continuity by œsophago-gastrostomy has become an established surgical procedure, the mortality rate for growths in the lower part of the œsophagus being well under 10% in the best clinics (Sweet, 1948). The operation is, however, more severe and the technique more exacting for growths in the middle and upper thirds of the œsophagus, and here the general mortality rate is still not low. Nevertheless, without surgery the patient's fate is so wretched that excision should be attempted. Even as a palliative measure the operation has immense value.

The patient is often in a state of advanced malnutrition and presents interesting problems in pre-operative preparation, maintenance during operation, and post-operative care. Successful surgery largely depends on understanding and grappling with these problems; the purely technical manœuvres to be mastered by the surgeon are not numerous. Full details of current techniques for removing growths high in the œsophagus need not be given here. My purpose is rather to underline some of the general problems and difficulties encountered in the management of these patients.

The following case is illustrative.

A.T., a married woman, aged forty-six years, was admitted to the Prince Henry Hospital on July 18, 1950, with a history of difficulty in swallowing and regurgitation for four months. Clinical and radiological studies showed her to be suffering from (i) carcinoma of the œsophagus just below the aortic arch and (ii) Raynaud's disease. A notable feature of the history was severe pain in the back (centrally about mid-dorsal level) when she attempted to swallow food. She presented obvious wasting, her body weight being only 88 pounds, and there was persistent cyanosis of the ears, nose, fingers and toes. Full clinical examination showed no evidence of distant metastasis. Blood examinations resulted as follows: on July 20 the hæmoglobin value was 8·96 grammes *per centum*, and the white blood cells numbered 56,000 per cubic millimetre with 96% of polymorphonuclear cells; on July 24 the hæmoglobin value was 8·68 grammes *per centum*, and the white blood cells numbered 53,800 per cubic millimetre with 97% of polymorphonuclear cells. The anæmia and probable reduction in circulating blood volume were corrected by means of three fresh blood transfusions, each of one litre, during the six days prior to operation, but the neutrophile cell count remained unchanged.

The patient was given a fluid diet, rich in carbohydrates and protein, thus ensuring a good general level of hydration and nutrition. The caloric intake during the ten days prior to operation averaged nearly 4000. On August 10 a left thoraco-abdominal incision was made under anæsthesia ("Pentothal", curare, nitrous oxide and oxygen) administered by Dr. T. A. K. Kennedy. The technique followed was similar to that of Garlock (1944) and Sweet (1946); only special points need to be mentioned here. The whole of the seventh rib and one inch of the posterior ends of the sixth, fifth and fourth ribs were excised. Free access is thus obtained inside the chest to the level of the third intercostal space. A hard mass partly neoplastic and partly inflammatory was found in the line of the œsophagus behind the left lung root. It was widely adherent to the right and left lung and pleura, the pericardium and the posterior aspect of the left lung root. To facilitate its safe removal the following course was adopted: (i) The lower part of the œsophagus was fully mobilized up to the growth. (ii) Inflammatory adhesions to the mediastinal surface of the left lung and to the posterior aspect of the pericardium and left lung root were carefully separated. (iii) The supraaortic part of the œsophagus was then fully mobilized, care being taken to dissect and preserve the recurrent laryngeal nerve and the thoracic duct. (iv) The uppermost three aortic intercostal arteries on both sides and the left bronchial artery were cleared and divided between ligatures near the aorta. (v) Finally some of the right mediastinal pleura and adherent right lung were resected, the latter being repaired with a running catgut suture.

The size of the mass prevented its being pulled through to the superior part of the mediastinum deep to the aortic arch. Therefore, when fully mobilized, the œsophagus was drawn down deep to the arch and divided as high as possible above the growth, leakage being prevented by a clamp below and by sucking out the upper end as the division proceeded. Two layers of interrupted silk (000) were used for the anastomosis. Before the anterior part of the mucosal layer was closed, a Ryle's tube was passed through the nose and guided under vision down into the pyloric antrum of the stomach. An underwater drain was brought out through the upper end of the chest wound. After completion of the operation ten ounces of blood were aspirated from the right pleural cavity.

I am grateful to Dr. Kennedy for excellent anæsthesia and maintenance during the operation; there was considerable hæmorrhage during separation of the growth, but the pulse and blood pressure remained almost constant throughout at seventy to ninety beats per minute, and 100 to 120 millimetres of mercury, respectively. Nearly 4·5 litres of blood were given during the procedure.

On the patient's return to the ward the air entry on both sides of the chest was checked regularly, and a slow intragastric drip of milk diluted with glucose water was given through the Ryle's tube; this was continued until removal of the tube on the sixth post-operative day.

The patient's convalescence was complicated, firstly, by atelectasis with superadded pulmonary infection, and then

by some leakage from the anastomosis. Despite every encouragement to cough, she tended to retain her sputum, which had become thick and tenacious forty-eight hours after the operation. On culture, it yielded a profuse growth of *Hæmophilus influenzæ*, hæmolytic streptococci and hæmolytic *Staphylococcus aureus*. These organisms were sensitive *in vitro* to a combination of penicillin, streptomycin and sulphadiazine. Therapy consisted of intramuscular administration of sulphadiazine one gramme and penicillin 400,000 units four-hourly, and streptomycin 0·5 gramme twice a day. Routine X-ray examinations of the chest with a portable machine were carried out twenty-four and seventy-two hours after operation. The later one showed considerable bilateral pneumonic consolidation of the lungs.

Oxygen was given intranasally throughout, but the decisive factor in her recovery was probably the repeated aspiration, through a Jacques's catheter, of both bronchi and the trachea. Her efforts at coughing remained feeble throughout, but this bronchial aspiration produced considerable quantities of purulent exudate. Whenever the exudate was allowed to accumulate in her respiratory passages she became cyanosed. Indeed, the combination of respiratory obstruction and Raynaud's disease resulted in an extraordinary degree of cyanosis. Frequent bronchial aspiration and continuous administration of oxygen, however, kept her a satisfactory colour most of the time. By the sixth post-operative day the lung infection was largely overcome and she had lost her dyspnœa.

When the Ryle's tube was removed, she took fluids by mouth. There was then slight leakage from the anastomosis through the upper end of the skin incision along the track of the underwater drain, which had been removed on the third day. This leakage at first required frequent changes of the dressing, but the wound quickly became dry and was practically healed fifteen days after operation. She was allowed out of bed on the eighth post-operative day. On August 22 her leucocyte count had fallen to 19,500 per cubic millimetre with 73% of polymorphonuclear cells. A report on an X-ray examination with swallowing of a barium bolus on October 5 stated that there was "slight delay in its passage through the anastomotic stoma and at the level of the diaphragm, but no organic lesion was indicated".

Pathological examination showed an ulcerated carcinoma involving the whole circumference of the œsophageal mucosa over a distance of five centimetres (in the fixed and hardened specimen) without obvious penetration of the œsophageal wall. Microscopically the growth was an anaplastic squamous carcinoma. No metastasis was found in several of the adjacent lymph glands sectioned.

After the operation the patient lost all her pain, dysphagia and regurgitation, but at the present time (June 26, 1951) there is slight stricture formation at the site of the anastomosis, which requires monthly bouginage.

## Points in the Management.

1. The importance of adequate supportive therapy in these patients, including blood transfusion before and during operation, needs no emphasis. Pre-operative transfusions restore the patient's depleted blood volume and widen his margin of safety against operative shock. During the operation blood should be given as it is lost, so that hypotension and collapse do not develop.

2. Careful radiological examination should also be carried out before operation to exclude mediastinal and lung involvement. In addition, endoscopy is necessary; the vocal cords are examined (finding a paralysed left vocal cord may save the patient from a fruitless thoracotomy), and the growth in the œsophagus is inspected. Its distance from the incisor teeth is measured and a biopsy is taken. If there are much stasis and œsophagitis above the growth, twice daily lavage with half-normal saline is indicated.

3. In the pre-operative preparation of these patients we use a fluid diet of six ounces (one cupful) of fortified milk mixture given hourly from 7 a.m. to 9 p.m. Each six ounces of mixture contain five ounces of milk and one ounce of cream, into which are stirred two tablespoonsful of powdered milk, the whole yielding approximately 300 Calories. At stated feeds this may be further enriched by the addition of eggs, lactose or sucrose. The flavour should be varied, it being given as a "porridge" mixture, creamy soup (cream of tomato, celery, potato, asparagus *et cetera*) or milk drink (coffee, cocoa, "Ovaltine" *et cetera*). Vitamin supplements, especially *B* and *C*, should be given. One of our patients with œsophageal obstruction achieved a daily intake of approximately 5000 Calories and gained ten pounds in fourteen days. Two to three weeks on this diet ensure that the patient goes into the operation well hydrated and without pronounced protein or carbohydrate depletion. The fluid balance should be carefully recorded for three days before operation and subsequently. If the pre-operative feeds are not taken well it may be advantageous to intubate the patient and give the mixture as a continuous intragastric drip.

4. Major gastro-œsophageal resections should be performed under adequate "antibiotic cover". If the patient has sputum, our practice is to prepare a culture from it and to determine the sensitivity of the organisms grown. If bronchitis is present, pre-operative penicillin inhalations may be helpful. A distinction should be made between the use of penicillin and allied agents as a prophylaxis, when the dosage may be small, and their therapeutic use in developing infections, when it should be large, as in the foregoing case.

5. Gentle handling of the tissues and meticulous suturing throughout are necessary for good results. I believe that it is useful to bring the underwater drainage tube out opposite the anastomosis, so that if leakage occurs there is a ready track to the exterior. Fortunately, with careful suturing such leakage is uncommon, but the manœuvre mentioned proved similarly helpful in one of my earlier cases.

6. Skilful anæsthesia and controlled respiration are, of course, essentials. During most of the operation deep anæsthesia is not required, since the surgeon is working in the chest with the ribs retracted mechanically and he has not to contend with the pull of the abdominal muscles. However, the arterial blood must be kept fully oxygenated throughout, and the left lung should be reinflated periodically.

7. The technique of continuous intragastric drip administration with a Ryle's tube passed through the anastomotic stoma has, I believe, advantages. It facilitates maintenance of the patient's fluid and caloric requirements, and having the tube *in situ* is helpful should gastro-duodenal ileus develop. It has been stated that such an indwelling tube may cause ulceration and dehiscence of the anastomosis, but the evidence in favour of this is not convincing, and it may be emphasized that a Ryle's tube of fine calibre is used.

8. Perhaps the most important single point in post-operative management is supervision of cardio-respiratory function. The right pleura is aspirated at the end of the operation, and the air entry to both lungs is repeatedly checked in the first few days. Routine X-ray pictures are taken with a portable machine on the first and third days after operation. The patient should be encouraged to cough from the beginning. This is best achieved by the patient sitting fully upright and with the chest supported above and below the incision. Coughing exercises should be practised before operation, especially in elderly subjects. The technique of tracheal and bronchial aspiration through a catheter passed under local anæsthesia should have been mastered by responsible members of the resident medical staff. If lung infection develops, the appropriate antibiotics are given in massive doses.

9. Finally, as a group, these patients are very wasted, and expert nursing care is necessary if sacral bed sores are to be avoided.

## Summary.

Some aspects of the pre-operative, operative and post-operative management of patients undergoing œsophagectomy for carcinoma are described. An illustrative case is reported. The patient, a woman aged forty-six years, presented with both Raynaud's disease and an extensive carcinoma of the mid-thoracic part of the œsophagus, associated with much local inflammatory change.

## Acknowledgements.

I am grateful to Miss D. M. Drake, of the Unit of Clinical Investigation, Royal North Shore Hospital of Sydney, for help in the preparation of this paper.

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## ENDEMIC LEPTOSPIROSIS IN VICTORIA.


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FROM 1935 onwards reports on bovine leptospirosis were made by Russian authors (Michin and Azinov, 1935; Terskikh, 1940; Semskow, 1941). Johnson in 1942 noted the presence of leptospiral antibodies in the serum of Queensland cattle. Further reports from America (Jungherr, 1944; Marsh, 1945; Mathews, 1946; Sutherland and Morrill, 1948; Baker and Little, 1948), from Palestine (Bernkopf *et alii*, 1947), and from Australia (Sutherland *et alii*, 1949) soon appeared. Published evidence of leptospirosis in Australian cattle still appears to be limited to the State of Queensland, but there is reason to believe that the same disease occurs in Western Australia (Mahaffey *et alii*, 1942).

It was observed by the senior author that the Victorian bovine disease, which occurs in the Gippsland district and which is known as "redwater", bore a close resemblance to the leptospiral bovine disease of Queensland (Sutherland *et alii*, 1949; Sutherland, 1950). Samples of serum from convalescent animals were forwarded to Professor E. Ford, of the School of Public Health and Tropical Medicine, Sydney. A significant number of these samples showed high antibody titres when tested with *Leptospira pomona*. These results, together with the presence of isolated cases of an influenza-like illness in Gippsland dairy farmers, prompted the present investigation.

In order to confirm the indirect evidence afforded by serological tests and to obtain more direct evidence of the nature of the disease, we felt that a prime object of the investigation should be the isolation of leptospiræ from diseased animals and, if possible, from man. For this reason stocks of guinea-pigs were carried on all field surveys, and appropriate specimens were inoculated into guinea-pigs with the minimum of delay.

### Materials and Methods.

Fletcher's semi-solid medium (Mackie and McCartney, 1948) and Korthof's fluid medium (van Thiel, 1948) were used for stock cultures, and in attempts to isolate leptospiræ from animal tissues. Incubation was carried out in the 30° C. incubator.

Serological tests were carried out by the technique of Davidson, Campbell, Rae and Smith (1934). This is a microscopic agglutination-lysis test with live cultures.

Attempts were made to isolate leptospiræ from the blood of sick animals, from the ground-up liver or kidney of recently dead calves, and from the urine of convalescent animals or man. Since it was seldom possible in the field to collect these specimens with due regard for aseptic precautions, direct in-vitro cultivation was not attempted. The selected material was inoculated intraperitoneally into two guinea-pigs, which were delivered to the laboratory within three days. Records of the rectal temperatures of these guinea-pigs were kept. When there was a rise in temperature one animal was sacrificed and examined for typical appearances such as "butterfly" hæmorrhages in the lungs. Otherwise both animals were killed at least fourteen days later, and an aseptic post-mortem examination was conducted. The guinea-pig sera were tested for antibodies, and cultures in Fletcher's and Korthof's media were made from the emulsified liver and kidney.

[1] Assisted by a grant from The National Health and Medical Research Council.

### Investigation of the Disease in Cattle.

The bovine disease is characterized by hæmoglobinuria and hæmoglobinæmia. Farmers may miss the diagnosis if they fail to observe the "redwater", which may last for one or two days only. Abnormal milk, especially the appearance of blood in the milk, has been noted by some observers (Baker and Little, 1948; Bernkopf *et alii*, 1947), but has not been a feature in our cases. Cows usually recover, but the mortality of the disease in calves is quite high.

A total of 42 east Gippsland cows and calves with suspected leptospirosis were investigated, and a diagnosis was made of *Leptospira pomona* infection in 25 animals. These results are summarized in Table I.

TABLE I.

*Investigation of 42 Cows and Calves with Diagnosis of Suspected Leptospirosis.*

| Type of Investigation. | Laboratory Findings. | | |
|---|---|---|---|
| | Indicative of *Leptospira pomona* Infection. | Negative. | Doubtful. |
| Serological tests .. .. .. | 15 | 8 | 2 |
| Serological tests *plus* evidence of infection in guinea-pigs .. | 6 | 1 | 0 |
| Evidence of infection in guinea-pigs .. .. .. .. | 4 | 5 | 1 |
| Total .. .. .. | 25 | 14 | 3 |

Since normal guinea-pigs from our laboratory stock failed to show *Leptospira pomona* agglutinins, it has been assumed that such agglutinins in guinea-pigs, fourteen days after inoculation with material from a suspected case of leptospirosis, were indicative of *Leptospira pomona* infection. This assumption has been confirmed in some instances by the cultivation of *Leptospira pomona* from the ground-up liver or kidney.

Table I indicates that in 15 cows or calves the diagnosis depended on significant titres to *Leptospira pomona* in the agglutination-lysis test, and in an additional 10 cows or calves the diagnosis was supplemented by, or depended upon, evidence of infection in the inoculated guinea-pigs. In four of the latter 10 cows or calves the infecting *Leptospira pomona* strain was isolated from guinea-pig tissues. The three laboratory findings listed as doubtful in Table I comprise two instances in which the *Leptospira pomona* titre was 1:40 and one instance in which the post-mortem appearances of inoculated guinea-pigs were suggestive of leptospirosis, but the guinea-pig sera were not tested.

### Investigation of the Disease in Man.

Human *Leptospira pomona* infection is manifest as an illness which clinically resembles influenza. A good account of the clinical syndrome has recently been given by Johnson (1950). Headache, fever and muscle pains are the prominent features, but meningeal symptoms may occur. The fever lasts for five to seven days, and a febrile

relapse is not uncommon. Jaundice is exceptional. The illness runs a benign course. The incubation period has been stated as seven to fourteen days, but may be shorter (Frey, 1948).

Forty-one persons associated with dairy farms were investigated. These persons fall readily into two groups. Sixteen persons had a history of suggestive illness, recent or remote, while 25 persons had no history of an illness suggestive of leptospirosis. The results of the investigation are summarized in Table II.

TABLE II.

*Investigation of 41 Dairy Farm Personnel.*

| Group. | | Serological Tests. | |
|---|---|---|---|
| | | Indicative of *Leptospira pomona* Infection. | Negative. |
| 16 patients with history of illness. | Nine examined with active disease. | 5[1] | 4 |
| | Seven reviewed in retrospect. | 4 | 3 |
| 25 patients without history of illness. | Workers on "redwater" farms. | 0 | 25 |

[1] A rise in antibody titre was demonstrated in successive "bleeds" from two of these patients.

It will be seen from Table II that nine of the 16 patients who suffered a suggestive illness gave evidence of *Leptospira pomona* infection, whereas the results of tests were uniformly negative on the group of 25 persons without history of such an illness. Of the seven patients reviewed in retrospect, the illness regarded as leptospirosis took place several months before serological tests were made, so that isolation of leptospiræ was not attempted. With seven of the nine patients with active disease attempts to isolate leptospiræ by guinea-pig inoculation were carried out.

With one of these patients the attempt was successful.

### Report of Case.[1]

L.V. was a boy, aged six years, who frequently played barefooted in the muddy surroundings of the cow and pig sheds on the farm where he lived. He developed a febrile illness characterized by headache, malaise and pains in the limbs. Headache was severe and lasted for ten days. The fever lasted for four or five days. Jaundice did not develop. On the ninth day from onset investigations were instituted. A sample of serum was taken and showed a *Leptospira pomona* titre of 1:20. Urine was collected aseptically and inoculated into two guinea-pigs.

Both guinea-pigs developed a febrile reaction seven days later. One guinea-pig was killed at the height of the febrile episode, when typical post-mortem appearances were seen, and the guinea-pig serum reacted to *Leptospira pomona* at a 1:20 dilution. The other guinea-pig, killed seventeen days after inoculation and four days after its temperature had returned to normal, had a serum antibody titre of 1:320 to *Leptospira pomona* and gave a growth of *Leptospira pomona* from its kidney tissue.

The patient had an uneventful convalescence. A further sample of serum was obtained forty days after the onset of his illness. The *Leptospira pomona* titre was now 1:160, and there was no reaction to *Leptospira canicola* or to other leptospira strains which have been recorded in Australia, namely, *Leptospira icterohæmorrhagiæ*, *Leptospira australis A*, *Leptospira australis B* and *Leptospira mitis*.

It is not unlikely that this patient illustrates one of the known routes of infection, namely, penetration of leptospiræ through a water-sodden skin.

### Investigation of Leptospirosis in Swine.

Bovine strains of leptospiræ have not been found in rats (Bernkopf *et alii*, 1948), which are the reservoir for *Leptospira icterohæmorrhagiæ* in classical Weil's disease, but a high *Leptospira pomona* infection rate has been observed in healthy swine (Collier, 1948; Roch and Mach, 1947). Reports by Johnson (1942, 1943) indicate that *Leptospira pomona* and *Leptospira mitis* infections occur in Queensland swine. In the present investigation it was observed that "redwater" broke out on one farm where no fresh cattle had been added to the herd for a long time, but a purchase of pigs had been made a short time previously. A serological investigation of swine on various east Gippsland "redwater" farms was therefore carried out, with the results set out in Table III.

TABLE III.

*Serological Tests on Swine.*

| Farm. | Number of Specimens of Swine Serum Tested. | Positive Reactors. | |
|---|---|---|---|
| | | *Leptospira pomona.* | *Leptospira mitis.* |
| C.C. .. | 8 | 1 | 6 |
| C.R. .. | 9 | 3 | 4 |
| S.C. .. | 7 | 4 | 2 |
| L.V. .. | 8 | 8 | 0 |
| H.B. .. | 6 | 0 | 0 |
| H.L. .. | 7 | 2 | 0 |
| Total .. | 45 | 18 | 12 |
| Grand total .. .. | | 30/45 | |

Table III indicates that not only is there a high infection rate amongst swine on "redwater" farms, but two different serological types of leptospiræ are concerned. There were instances of cross-reaction between *Leptospira pomona* and *Leptospira mitis*, but swine sera, in the main, reacted only to one or other of these strains. Of the six farms from which swine sera were collected, it is observed that on one farm no reactors were found, on five farms there were *Leptospira pomona* reactors, and on three of the latter *Leptospira mitis* reactors were also found. On one farm (C.C.) *Leptospira mitis* reactors outnumbered *Leptospira pomona* reactors. These observations are being extended in an endeavour to find *Leptospira mitis* infections amongst cattle (for example, on farm C.C.) and to isolate both leptospiral strains from swine.

Leptospiral infection in swine appears to be symptomless in the majority of cases, although clinical illness may result (Wagener, 1942; Schmid and Giovanella, 1947). Of the several hundred pigs on the various "redwater" farms only two pigs had symptoms suggestive of leptospirosis. It seems probable, therefore, that *Leptospira pomona* possesses a descending order of pathogenicity towards calves, adult cattle, man and swine.

### Investigation of Men Connected with the Meat Industry.

There were available a number of specimens of serum collected in the course of a brucellosis survey (W.J.S.), taken from men connected with the meat industry in various parts of Victoria—abattoir workers, butchers and meat process workers. These specimens represented a sample of an occupational group which might well be expected to furnish evidence of leptospirosis if such existed in other parts of Victoria. These specimens, along with a control group, representing specimens of serum submitted for Wassermann test and supplied by courtesy of Dr. M. Wilson, of the Public Health Laboratory, were tested with the two leptospiral strains found in Gippsland, with the results set out in Table IV.

These reactions appear to be specific, and it is reasonable to assume that 13% of this group of meat workers had suffered a *Leptospira pomona* infection in the course of their occupational duties. Since no record had been kept of the medical histories of the men concerned, it was not possible to relate their leptospiral infection to any illness they may have suffered.

[1] Notes supplied by courtesy of Dr. H. L. McCay, of Orbost.

In view of the fact that a high proportion of the Gippsland swine gave evidence of *Leptospira pomona* infection, it was to be expected that men working whole-time with swine, that is, bacon factory workers, would show a higher incidence of leptospirosis than other meat workers who handled swine less frequently. Table V shows that bacon factory workers did suffer a higher infection rate than other meat workers.

TABLE IV.

*Serological Tests on Meat Workers.*

| Group. | Number of Specimens of Serum Tested. | Number of Positive Reactors. |
|---|---|---|
| Serum from meat workers .. | 305 | 40[1] (13%) |
| Control series .. .. | 200 | 0 |

[1] All of these were *Leptospira pomona* reactors. One specimen of serum also reacted to *Leptospira mitis*.

From further analysis of occupation within the meat industry in relation to *Leptospira pomona* infection, it is seen that slaughtermen, as would be expected, had a significantly higher incidence of infection than had other meat workers. This analysis is shown in Table VI.

TABLE V.

*Distribution of Positive Reactions amongst 305 Meat Workers.*

| Bacon Factories. | Abattoirs. | Shop Butchers. | Meat Processing. |
|---|---|---|---|
| 16/49 | 17/139 | 7/91 | 0/26 |

Total 40/305 (13%)

Of the occupations listed in Table VI only the slaughtermen constantly handled carcasses which still retained their animal heat. It is interesting to note that 49 slaughtermen handled pigs all or part of their time, and that 17 of these men had been infected with *Leptospira pomona*.

TABLE VI.

*Relation of Occupation within he Meat In dustry to Leptospiral Infection.*

| Occupation. | Number of Specimens of Serum Tested. | Positive Reactors. |
|---|---|---|
| Slaughtermen .. .. | 107 | 27 (25%) |
| Shop butchers .. .. | 91 | 7 (8%) |
| Labourers .. .. | 32 | 2 |
| Meat packers .. .. | 23 | 2 |
| Meat by-products .. | 16 | 1 |
| Gut scrapers .. .. | 14 | 0 |
| Hides and offal .. | 12 | 0 |
| Meat inspectors .. | 10 | 1 |

## Discussion.

The present communication indicates that leptospirosis occurs as an endemic disease in Victoria. The important infecting strain is *Leptospira pomona*, which was first isolated, from a case of human leptospirosis, in Queensland (Clayton and Derrick, 1937; Derrick, 1942), but has since been found in many other parts of the world. In Victoria, it is interesting to note, the bovine disease was first to attract attention and indicated the possibility that human infection might also occur. The human illness is not particularly characteristic and might otherwise have escaped detection for much longer.

It has been demonstrated by various workers that swine are an important reservoir of *Leptospira pomona* infection, and evidence is presented to show that the same conditions prevail in Victoria. That the reservoir of infection in cattle is potentially less dangerous to man than the swine reservoir is shown by the findings of leptospirosis in Victorian meat workers.

## Summary.

1. Leptospirosis exists in Victoria as a disease of cattle and of humans in certain occupational groups, namely, the dairying industry and the meat trade.

2. Such infections have been caused by *Leptospira pomona*.

3. An important reservoir of *Leptospira pomona* infection exists in swine, and there is serological evidence that Victorian swine are also carriers of *Leptospira mitis*.

## Acknowledgements.

Professor E. Ford, of the School of Public Health and Tropical Medicine, Sydney, was kind enough to supply us with *Leptospira* strains and sera, together with much helpful advice, so that the laboratory work for this investigation could be carried out in Melbourne. We are also indebted to Dr. E. V. Keogh for advice and criticism, to Dr. H. E. Albiston, and to the various doctors in Gippsland, especially to Dr. J. E. D. Lane and Dr. H. L. McCay, who have notified us of cases and aided in these investigations. Much technical help in the laboratory was given by Miss Coralie Potter.

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# Reports of Cases.

## TWO CASES OF HUMAN INFECTION WITH LEPTOSPIRA POMONA IN VICTORIA.

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FIVE species of leptospires are known to cause human leptospiral infection in Australia. As Johnson (1950) has suggested, infections due to these organisms may be grouped conveniently in three categories:

1. Classical Weil's disease caused by *Leptospira icterohæmorrhagiæ*. Reported cases of infection with this strain are relatively few in number. They have occurred in urban areas, chiefly in Queensland, but also in New South Wales and Victoria. Animal reservoirs of infection are the rats, *Rattus norvegicus* and *Rattus rattus*.

2. Canefields leptospiroses, in which the bacterial agents are *Leptospira australis A* and *Leptospira australis B* (the incomplete biotype of *Leptospira pyrogenes*). Infections with these strains have been reported in North Queensland only. The endemic area includes the cane-growing country extending from Mossman in the north to Ingham in the south. The main animal reservoir of both strains is the field rat, *Rattus conatus*.

3. Mild leptospiroses caused by *Leptospira pomona* and *Leptospira mitis*. Infections with *Leptospira pomona* have been reported in Queensland, chiefly southern Queensland, and in northern New South Wales, whilst Johnson (1950) has made reference also to the occurrence of pomona-type leptospirosis in Perth.

*Leptospira pomona* is endemic in Australian pigs and cattle (Johnson, 1950). This strain has been isolated by Sutherland, Simmons and Kenny (1949) in Queensland, from calves suffering from bovine leptospirosis, known previously as "red water" of calves. More recently, infection of calves in the south-west of Western Australia has been recorded by Peterson (1951). A serological survey of Australian dogs for leptospiral infection, as yet unpublished, carried out at the School of Public Health and Tropical Medicine, demonstrated anti-pomona agglutinins of significant titre in Victorian dogs.

It would appear that *Leptospira pomona* can be transmitted to man from animal reservoirs in two ways: (i) by direct contact of persons with infected animals—for example, butchers, dairy farmers and veterinary workers—and (ii) by the medium of water or mud contaminated with leptospires. It is significant that, in rural areas, leptospirosis occurs most frequently after rain.

Johnson (1950) records the prominent signs and symptoms of pomona-type leptospirosis as follows: headache; muscular pains; meningeal involvement, frequently transient; gastro-intestinal disturbance; prostration; renal involvement; eye complications—for example, photophobia, iritis or iridocyclitis; pulmonary signs; and, in some cases, exanthemata or arthritis. Jaundice occurs rarely, and liver damage would appear to be much less common than in classical Weil's disease. In the majority of cases pyrexia lasts from seven to ten days, but it may persist for as long as thirty-five days, or terminate after three days.

In most cases the onset of the disease is sudden, with the prompt appearance of pyrexia, headache and general malaise. Less frequently the early signs of infection are more insidious in character. Although the illness may be severe, the prognosis is excellent.

### Clinical Records.

CASE I.—R.G., aged forty-seven years, a share farmer on a dairying property, came under the observation of the senior author on April 6, 1950. He complained of fever and malaise for three or four days and had been confined to bed for twenty-four hours. At the time of examination, his chief complaints were generalized muscular pains involving mainly the back and limbs; headache; weakness; photophobia; and some pain in the chest with slight cough.

The patient's temperature was 102° F. There was moderate tenderness of the muscles of his neck and back with some neck rigidity. Scattered moist sounds were detected over the lower part of his right lung. No other abnormality was found. The clinical signs detected in the patient were disproportionate with the severity of his illness.

Penicillin was administered intramuscularly in doses of 50,000 units every six hours. After forty-eight hours the patient's temperature fell to normal and pyrexia did not recur. However, convalescence was slow, and he did not feel able to resume work for a further ten weeks.

CASE II.—L.G., aged sixteen years, a son of the patient in Case I, suffered similar symptoms at the same time as his father. His clinical condition was milder, and his temperature returned to normal in a few days. An analgesic mixture was given, but penicillin was not exhibited in this case.

### Comment.

Both patients were thought to have suffered moderately severe attacks of influenza. However, in September, 1950, Mr. N. A. M. Wellington, Veterinary Officer, Department of Agriculture, Victoria, brought to notice the incidence of bovine leptospirosis on the property where the patients were employed. Suspecting leptospiral infection, he had submitted specimens of serum from cattle to the School of Public Health and Tropical Medicine for serological investigation. Examination of six of these specimens revealed anti-pomona agglutinins, in titres ranging from 1:30 to 1:3000.

The provisional diagnosis of influenza was reviewed in the light of this information. Anti-pomona agglutinins were demonstrated in the serum of both patients, in each case to a titre of 1:300. No reactions were detected with the antigens of other Australian strains or with *Leptospira canicola*. The serological method employed was the agglutination-lysis technique described by Schüffner and Mochtar (1927). It is considered that the titres of agglutination-lysis recorded are adequate for a diagnosis of leptospiral infection, particularly in view of the clinical histories and the incidence of bovine leptospirosis in the immediate environment of the patients.

### Discussion.

Prior to investigation of the two cases described, human infection with *Leptospira pomona* had not been identified in Victoria, and it is thought desirable to place them on record.

It is a matter of epidemiological interest that widespread flooding occurred in the Gippsland district during April, 1950, and the ground surrounding the dairy shed in which the patients worked, was a sea of mud for a distance of about fifty yards in all directions. Consequently, the possibility of skin contamination with leptospires deposited by infected animals was considerable.

### Acknowledgements.

Thanks are due to Mr. N. A. M. Wellington, Veterinary Officer, Department of Agriculture, Victoria, for his cooperation, to Professor Edward Ford, Director, School of Public

Health and Tropical Medicine, for his interest in the work, and to the Director-General of Health, Canberra, for permission to publish this paper.

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# Reviews.

## A YEAR BOOK OF ORTHOPÆDICS AND TRAUMATIC SURGERY.

"THE 1950 Year Book of Orthopedics and Traumatic Surgery" is a relatively slim volume, but it covers much useful ground.[1] The editor, Edward L. Compere, has drawn his material from journals received between November, 1949, and November, 1950, and has enhanced its value by a generous policy in the inclusion of illustrations. The world-wide range of journals covered is notable, British and European work being particularly well represented. Australian authors whose papers are abstracted include Cyril H. Chambers, B. Keon-Cohen and Thomas King, of Melbourne, and L. J. Woodland, of Sydney. The abstracts, with very occasional comments by the editor, are arranged into chapters on poliomyelitis, congenital deformities, the embryology, physiology and anatomy of the skeletal system, the epiphyses, osteomyelitis and other infections, tumours, cysts and fibrodysplasia, arthritis and rheumatism, fractures, the spine and pelvis, the neck, shoulder and arm, the hand, the hip, leg and ankle, the foot and ankle, amputations and prostheses, surgical technique, instruments, appliances and bone banks, and miscellaneous topics. In his introductory special article on progress in orthopædic surgery between 1940 and 1950, the editor points out that this progress has been made more slowly than in some other fields of medicine. Nevertheless, his review and the accounts of current work in the present volume show worthwhile advances. This volume should be a great aid to orthopædic and general surgeons, pædiatricians and others who are concerned to keep themselves abreast of such advances.

## A YEAR BOOK OF NEUROLOGY, PSYCHIATRY AND NEUROSURGERY.

EACH of the three sections of "The 1950 Year Book of Neurology, Psychiatry and Neurosurgery" is self-contained, with its own editor, and following the practice in this year's series of Year Books, each editor contributes a review of progress in his own subject from 1940 to 1950.[2] Notable advances have occurred in all three fields. Current work also is important, and the editors have abstracted from journals received by them between November, 1949, and October, 1950, a variety of useful and interesting material. The plan of the section on neurology is little changed, containing chapters on anatomy and physiology, pathology, trauma, infectious diseases (meningitis, encephalitis and myelitis, poliomyelitis), degenerative diseases, cerebrovascular disorders, convulsive disorders, neuropathies and neuralgia, and diagnostic and therapeutic methods. Reference is made to a paper on cerebral lesions due to intracranial aneurysms by E. Graeme Robertson, of Melbourne, and to papers on "Diparcol" by W. Lister Reid and by R. B. Pilcher, both of Sydney. A feature of this section is the heading of most chapters with a brief editorial discussion on the related subject matter.

The section on psychiatry has chapters on general topics, child psychiatry, schizophrenia, affective disorders and miscellaneous reactions, organic disorders and toxic reactions, psychoneuroses and psychosomatic disorders, and therapy (psychotherapy, insulin shock therapy, electric shock, psychosurgery, miscellaneous therapeutic procedures). Editorial comment is sparse and brief.

In the section on neurosurgery the chapters are devoted to intracranial tumours, epilepsy, leucotomy, motor disorders, pain, suppuration, hæmorrhage, electroencephalography, cerebral circulation, radiology, intraspinal tumours, herniated disks, malformations, peripheral nerves, cranial nerves, sympathetic nerves and miscellaneous topics. A paper is included by Thomas King, of Melbourne, on the treatment of traumatic ulnar neuritis.

A wide range of journals has been covered to fill the approximately two hundred pages of each section, and apart from the specialists for whom the book is primarily designed there will be few medical practitioners to whom some parts at least do not appeal.

## MALIGNANT DISEASE OF THE FEMALE GENITAL TRACT.

WRITTEN principally for clinical gynæcologists, Stanley Way's monograph[1] dealing with malignant disease of the female genital tract treats this subject from every aspect.

The book commences with a discussion of carcinoma of the vulva and the other conditions are dealt with *seriatim* in ascending order, appropriately one-third of the volume being devoted to carcinoma of the cervix. The author maintains that the treatment of vulval carcinoma is essentially surgical, but to be effective it must be extremely radical, including lymphadenectomy in every instance. With regard to cervical cancer, those cases responding favourably to radium, as assessed by serial biopsies, can, he states, be dealt with better by irradiation than by surgery and with a lower mortality. On the other hand, in those cases responding unfavourably, the prognosis is inevitably bad, but surgery holds out the most hope. More patients will be saved by earlier diagnosis, more attention to detail and individualization of treatment, and more experience of the team. The remainder of the book is devoted to a consideration of carcinoma of the *corpus uteri*, the tubes and ovaries, sarcomatous lesions of the genital tract, chorionepithelioma and melanoma; it concludes with a chapter concerning the problems that arise when pregnancy is complicated by gynæcological malignant disease.

The author impresses the reader as a person of very exacting standards, and as one writing with authority born of experience. Anything savouring of speciousness is very ruthlessly dealt with and old concepts that cannot be reconciled with his own personal experience are subjected to a very critical, sometimes devastating, scrutiny. We feel also that in his own work he is equally uncompromising. Malignant disease of the female genital tract provides a focal point for the work of the gynæcologist, radiotherapist and pathologist, and this concise yet comprehensive survey can be warmly recommended to all practitioners in these specialties.

## AN INTRODUCTION TO PATHOLOGY.

"AN INTRODUCTION TO PATHOLOGY" by G. Payling Wright, Professor of Pathology at Guy's Hospital Medical School, is a distinguished and remarkable book to which it is difficult to do justice in a brief review.[2] In his preface, the author observes that there is a tendency in the training of students for an increasing emphasis to be placed on the practical applications of pathology to diagnosis. There is,

[1] "The 1950 Year Book of Orthopedics and Traumatic Surgery (November, 1949-November, 1950)", edited by Edward L. Compere, M.D., F.A.C.S.; 1951. Chicago: The Year Book Publishers, Incorporated. 7¾" × 5¼", pp. 388, with 264 figures. Price: $5.00.

[2] "The 1950 Year Book of Neurology, Psychiatry and Neurosurgery" (November, 1949-October, 1950); Neurology, edited by Roland P. Mackay, M.D.; Psychiatry, edited by Nolan D. C. Lewis, M.D.; Neurosurgery, edited by Percival Bailey, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 8" × 5¼", pp. 628, with 121 illustrations. Price: $5.00.

[1] "Malignant Disease of the Female Genital Tract", by Stanley Way, M.R.C.O.G.; 1951. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 278, with 38 illustrations. Price: 24s.

[2] "An Introduction to Pathology", by G. Payling Wright, D.M., F.R.C.P.; 1950. London, New York, Toronto: Longmans Green and Company. 8¾" × 6", pp. 588, with many illustrations. Price: 49s. 6d.

he believes, a serious danger that the practical aspects may be over-emphasized, to the detriment of the student's knowledge and grasp of fundamental principles. This book is written therefore as an approach to general pathology; and it is written from two essentially dynamic points of view. Firstly, emphasis is placed on ætiology—the scientific study of the causation of disease—and secondly, the book is intended as an exposition of the principles of pathology in terms of the more fundamental sciences of biology and physiology. It is based on Professor Wright's undergraduate teaching course, but it is not a text-book of pathology in the ordinary sense. One feels, at first reading, that this is a book for the student of unusual ability, or for the post-graduate who will find here an excellent revision and synthesis of the knowledge he has been acquiring over the years, but that it is somewhat beyond the grasp of the average undergraduate. On reflection, however, one realizes that perhaps it depends on how undergraduates are taught. In the preface, it is pointed out that a book on general pathology can never take the place of personal experience in the post-mortem room, the museum and the practical classroom. But undergraduates who have already acquired a thorough groundwork of practical knowledge may perhaps welcome the intellectual background provided by a book such as this, in which the facts they have been learning are fitted into a plan and made intelligible and even beautiful just as the pieces of a jig-saw puzzle fall into place and make a coherent picture. Undoubtedly the author is setting himself and his students a high standard, and is anticipating the future. To quote his preface: "In a branch of knowledge that is advancing as rapidly as modern medicine, it is becoming increasingly difficult to draw any distinction between its academic and its practical aspects. It thus becomes more than ever desirable to provide medical students not only with a knowledge of current practice, but also with an intellectual background which will enable them to make effective use of the advances of the future." This book is extremely well written, though it is not easy to read. It calls for some intellectual effort; it is not to be skimmed; one must read every sentence. It is more concerned with pathology as "disturbed physiology" than with morphological descriptions; there are, however, many excellent illustrations, more than half of which are photomicrographs. Frequent reference is made to the problems of medicine and surgery and the introduction is followed by a chapter on "Ætiology: The Causes of Abnormalities and Disease".

Modern points of view on neoplasia are well described; three chapters are devoted to the ætiology of tumours and another to the precancerous state. The emphasis on ætiology gives a note of hopefulness and progress; behind the "pestilential vapours" of disease there is a "brave o'erhanging firmament". The author writes that: "Pathology is commonly defined as the 'Science of Disease', though its more lengthy description as the scientific study of 'any condition of the organism which limits life in either its powers, enjoyment or duration' is in many respects to be preferred." There sounds the positive note of challenge and optimism; it should not be a very far step from ætiology to prevention.

## Books Received.

*[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]*

*"Le syndrome d'induction sensorimotrice dans les troubles de l'equilibre"*, by L. Halpern; 1951. Paris: Masson et Cie. 9" × 5¾", pp. 104, with illustrations. Price: 400 fr.

The book deals with the author's researches on sensorimotor phenomena and their bearing on clinical practice and neurological investigation.

"Handbook of Medical Management", by Milton Chatton, A.B., M.D., Sheldon Margen, A.B., M.D., and Henry D. Brainerd, A.B., M.D.; Second Edition; 1951. Palo Alto, California: University Medical Publishers. 7" × 4¼", pp. 512, with a few illustrations. Price: $3.00.

This book, intended for the student and practitioner, indicates what should be done when the diagnosis has been established. It shows what should be done first and explains how chemical and other methods should be used. Eighteen chapters are devoted to different groups of diseases.

"The Doctor: His Career, His Business, His Human Relations", by Stanley R. Truman, M.D.; 1951. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6¼", pp. 158. Price: 32s. 3d.

Intended chiefly for the recent graduate, this book deals with professional problems and with the relationship and responsibility of the practitioner to the patient, the public and other practitioners.

"Disease in Infancy and Childhood", by Richard W. B. Ellis, O.B.E., M.A., M.D., F.R.C.P.; 1951. Edinburgh: E. and S. Livingstone, Limited. 10" × 6", pp. 704, with 300 illustrations, some of them coloured. Price: 42s.

The book is intended "to emphasize those features of the young and growing individual which will determine his response to the impact of disease processes".

"Scientific Thought in the Twentieth Century: An Authoritative Account of Fifty Years' Progress in Science", edited by Professor A. E. Heath, M.A.; 1951. London: Watts and Company. 8½" × 5½", pp. 404. Price: 42s.

Fifteen chapters by fifteen different authors.

"Primary Carcinoma of the Liver: A Study in Incidence, Clinical Manifestations, Pathology and Ætiology", by Charles Berman, M.D., B.Ch. (Rand.), with a foreword by Sir Ernest Kennaway, M.D. (Oxon.), D.Sc. (London), F.R.S., F.R.C.P., and an introduction by Henry Gluckman, M.R.C.S. (England), L.R.C.P. (London); 1951. London: H. K. Lewis and Company, Limited. 10" × 7½", pp. 180, with 83 illustrations. Price: 35s.

Based chiefly on work done among the Bantu.

"Addendum 1951 to the British Pharmacopœia 1948", published under the direction of the General Medical Council pursuant to the *Medical Council Act*, 1862, and the *Medical Act*, 1950. London: Published for the General Medical Council by Constable and Company, Limited. 8½" × 5¾", pp. 132. Price: 17s. 6d.

This addendum is published in accordance with the provisions of the *Medical Act*, 1950, of Great Britain and will become official on September 1, 1951.

"The Medical Clinics of North America" (issued every two months); 1951. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. Nationwide Number, March, 1951, issue. 9" × 6", pp. 338, with 124 illustrations. Price: £7 5s. *per annum* (cloth binding) and £6 *per annum* (paper binding).

Consists of two parts. The first is a symposium on diseases of the skin; the second is a symposium in honour of William Sharp McCann and consists of a foreword and eleven articles on a variety of medical subjects.

"The 1950 Year Book of Dermatology and Syphilology (December, 1949-November, 1950)", edited by Marion B. Sulzberger, M.D., and Rudolf L. Baer, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 8" × 5¼", pp. 498, with 67 illustrations. Price: $5.00.

One of the "Practical Medicine Series" of year books.

"Growth and Development of Children", by Ernest H. Watson, M.D., and George H. Lowrey, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 9" × 6", pp. 260, with 54 figures. Price: $5.75.

Based on material gathered from many sources.

"Medical Treatment: Principles and Their Application", edited by Geoffrey Evans, M.D., F.R.C.P.; 1951. London: Butterworth and Company, Limited. 10" × 7", pp. 1490, with 51 figures. Price: 127s. 6d.

The object of the book is to reflect the present-day outlook on medical practice and to make generally known the treatment practised by its fifty-three contributors.

"The 1950 Year Book of Pathology and Clinical Pathology (January-December, 1950); Pathology, edited by Howard T. Karsner, M.D., LL.D., Clinical Pathology, edited by Arthur Hawley Sanford, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 8" × 5¼", pp. 456, with 157 illustrations. Price: $5.00.

One of the Practical Medicine Series of year books.

The Medical Journal of Australia

SATURDAY, JULY 7, 1951.

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*Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.*

## OCCUPATIONAL FACTORS IN THE ÆTIOLOGY OF PEPTIC ULCER.

THE psychological factor in the causation of peptic ulcer has been recognized for many years. Knowledge concerning a nervous factor ranges from the clinical recognition of the highly strung patient with peptic ulcer to the fact that gastric erosions can be produced experimentally by causing lesions in the central nervous system. The nervous tension manifested by men and women is often the result of occupations which they follow, but presumably there are folk who are psychologically disturbed because of the fact that they have not enough to occupy them. Many occupations come to mind which are likely to cause mental stress and worry. Medical practice in its concentrated form is one of them; the driving of motor omnibuses or lorries is another; the attendance on telephone switches can be most exacting; and the controlling of businesses in which heavy responsibility has to be taken and quick decisions must be made should not be forgotten. Of fundamental importance, of course, is the temperament of the worker. What is to one person like water on a duck's back may be to another a constant source of irritation and anxiety. In this regard the question of temperament might be discussed in detail, but this is not possible at the moment. The present object is to draw attention to a report recently issued in England by the Medical Research Council on occupational factors in the ætiology of gastric and duodenal ulcers.[1]

The investigation on which the report is based was sponsored by the Industrial Health Research Board of the Medical Research Council; it was facilitated by the appointment of a Peptic Ulcer Coordinating Committee, of which the late Professor J. A. Ryle was chairman, and Avery Jones and Doll, the two authors of the report, were members. At the outset it may be pointed out that the report fails to confirm some commonly held ideas and this is one reason why particular attention should be paid to it. There were in the United Kingdom three sources of information to enable the effect of occupational factors to be assessed—the Registrar-General's mortality statistics, hospital records, and insurance certificates and industrial sickness-absence records. As none of these was entirely satisfactory, it was decided to interview directly a number of men employed in various occupations and to make personal clinical diagnoses. The cooperation of a number of firms was obtained and a social worker interviewed selected groups of employees, asking whether they had ever had a peptic ulcer or had suffered from indigestion. The subjects were classified into three groups under three headings—"major dyspepsia", "minor dyspepsia" and "no dyspepsia". The subjects classified as having major dyspepsia were sent to be interviewed by one of the medical workers; and 10% of the subjects in the other two groups were sent to be interviewed as controls. Since only two of the 474 control subjects were found on being interviewed to have peptic ulcer, it is concluded that the method of selecting subjects for medical interview was reasonably reliable. Altogether, 6047 men and women were included in the survey and of these, 5951, or 98·4%, were interviewed by the social worker; 1302 were referred for a medical interview and 1284, or 98·6%, were seen. It was concluded that no appreciable error could have been introduced by the failure to interview the whole sample of the population covered by the survey. At the medical interview a diagnosis of ulcer was made if an ulcer had been discovered on radiological examination or at operation, or if hæmatemesis or gross melæna had occurred. If possible an X-ray examination was made if the history of the pain was suggestive of ulceration. This was not always possible, however, and in some instances the diagnosis had to be made on the history of the symptoms alone. Barium meal examinations were sometimes carried out and four ulcers were discovered in this way. These examinations showed that the clinical diagnosis was not exact, but the conclusion was that, short of mass barium meal studies of the whole population, it was not possible to obtain more accurate diagnosis. A total of 334 peptic ulcers was diagnosed, and of these, 69, or 21%, were presumptive ulcers diagnosed on the history of the pain alone. Among 4871 men 316 ulcers were diagnosed, an incidence of 6·5%; 18 ulcers were discovered among 1080 women, an incidence of 1·7%. It is pointed out that these rates cannot be applied directly to the general population because the sample surveyed was not representative of it. Only in regard to social class composition was the sample reasonably representative. When allowance is made for age and sex, and when smaller occupational and social class differences are neglected, the incidence of peptic ulcer in London is calculated as being 5·8% for men between the ages of 15 and 64 years, and 1·9% for women in the same age group.

After this somewhat lengthy but necessary introduction we may turn to the main topic of the report. One fact of primary importance is stated—that from the point of view of research it is unfortunate that men do not remain in the same occupation all their lives, and the question arises how far the incidence can be taken to reflect truly the characteristics of the occupations at the time of the

[1] "Occupational Factors in the Ætiology of Gastric and Duodenal Ulcers, with an Estimate of their Incidence in the General Population", by Richard Doll and F. Avery Jones, with the assistance of M. M. Buckatsch; Medical Research Council of the Privy Council, Special Report Series, Number 276; 1951. London: His Majesty's Stationery Office. 9¼" × 6", pp. 96. Price: 2s. 6d. net.

survey. A man may select a certain occupation because he has or has not an ulcer, but if no such selection has occurred, it should not matter that symptoms may have begun when the man was engaged on some occupation other than the one recorded at the time of the interview. In the present instance the possibility of differential selection of occupation cannot, we are told, be excluded. Of 316 men 30 had changed their occupation because they had ulcers; six had changed the type of shift worked; a further six had changed their place of employment without otherwise altering the nature of their occupation. To appreciate the results of the investigation in regard to occupation it will be helpful to note some of the observations made about age incidence, age at the apparent onset of ulcers and the differences in social status of the subjects. The incidence of peptic ulcer was found to vary considerably with age. In men the incidence increased to a maximum of 9·6% in the age group 45 to 54 years and then decreased. The incidence in women rose steadily with age to a maximum of 6·1% at ages over 55 years, but the numbers were so small that the observed incidence is not reliable. When gastric and duodenal ulcers were considered separately the maximum incidence of gastric ulcer was found to be in the decade from 45 to 54 years; the maximum incidence of duodenal ulcer was ten years earlier. From the age of onset of the observed ulcers and the number of years already lived by the persons surveyed at each age it was possible to calculate "the annual expectation of developing an ulcer". Contrary to what is generally thought, the expectation of developing a peptic ulcer was almost constant; it was at its maximum between the ages of 35 and 64 years, when the annual rate was of the order of 3·2 ulcers per thousand men. It is suggested that the idea that peptic ulcers develop oftener in young men is due to a failure to take into consideration the relative sizes of the populations at risk. Gastric and duodenal ulcers behave differently with regard to the age of onset; the expectation of developing a gastric ulcer is greatest between the ages of 35 and 64 years, while the expectation for duodenal ulcer is at a maximum and is constant between the ages of 20 and 64 years. The men interviewed were placed in one of five social classes, as follows: (i) leading professions, directors of companies *et cetera*; (ii) lesser professions, employers and managers in industry, transport, trade *et cetera*; (iii) skilled workers, including clerks and salesmen; (iv) semi-skilled workers; (v) unskilled workers. It was found that social class did not influence the incidence of duodenal ulcers. On the other hand, gastric ulcers presented a sharp gradient with social class. In classes (i) and (ii) the incidence was two-thirds less than that expected; in class (v) it was two-thirds more. The occupations with high incidence of peptic ulcer are discussed, as well as those with low and average incidences. High incidences were found among doctors and among certain unskilled workers. The high incidence among the former is attributed to the greater refinement of diagnosis and among the latter to the selection of sheltered employment by ulcer subjects. High incidences were also found among foremen and business executives. Here the excess of ulcers is considered significant only if it is regarded as justifiable to treat the two groups as a single entity. There seems to be no reason why they should not be so treated. A low incidence was found among agricultural workers and possibly among sedentary workers. Occupations with average incidences included bus conductors and drivers of motor vehicles. (Special care was taken to ensure inclusion of an adequate number of these.) Other large groups which showed incidences close to those expected were skilled engineering and electrical tradesmen, heavy manual workers, semi-skilled and unskilled and unclassified groups of workers in social classes (iii) and (iv). The contention that the high incidence among doctors is due to greater refinement of diagnosis is most interesting and probably there is some truth in the contention. If this is granted, however, it seems difficult to escape the conclusion that had the same refinement of diagnosis been available to other subjects, the incidence in their cases might have been higher. There is no doubt that a doctor would notice and take heed of his own melæna, while a non-medical person might not.

An analysis of factors which might be responsible for occupational differences is made. These include shift work, irregular meals, anxiety and other factors. Of these the most important is clearly anxiety. Shift work was found not to be of importance, and though there seemed to be an association between peptic ulcer and irregular meals, it was concluded not to be of major importance. In regard to anxiety over work it was found that its association with peptic ulcer was entirely confined to duodenal ulcer and that it was immaterial whether the ulcer was active or not. It is held, and justifiably, that the association is a real one. Of course, as the reader of the report is reminded, work is only one aspect of life which may give rise to anxiety, and the histories on which the report is based were superficial. Three psychological interpretations of the association of anxiety with duodenal ulcer are named as being in line with current thought—"that the duodenal subjects had more causes for worry or that their conditions were not unlike those of other men, but, having worrying personalities, they found the same conditions more trying, or that they had the type of personality which tended to result in their being employed in worrying positions". It is the last of these interpretations which the authors favour. They present arguments to support this view and they state that, whatever may finally prove to be the chain of causation, their findings strongly confirm the importance of psychosomatic factors in the ætiology of duodenal ulcer.

This report, which should be studied by every practitioner concerned with the fundamentals of disease of the stomach and duodenum, gives much food for thought. It does not herald any great advance; indeed its authors use the word iconoclastic about it. But procedures which can be so described are sometimes necessary. Even if many of our ideas on occupation and peptic ulcer are not correct, we still have to discover how mental processes can become linked with processes in the upper end of the alimentary tract. A man may not be able by taking thought to add a cubit unto his stature, but something allied to the taking of thought may upset his whole being and make stature of no account. The general conclusion—the need for further research on the subject—is obvious. The Medical Research Council, at the end of its introduction, states that the high prevalence of the disease, its claim on hospital beds and its unsolved ætiology all make it worthy of further intensive study.

# Current Comment.

## ITCH, "ITCHY SKIN" AND TICKLE.

A GOOD DEAL of interest has been taken in three phenomena that appear to be qualitatively similar—itch, tickle and the "itchy skin" phenomenon, in which an itching sensation is produced by stroking with the finger or a blunt object the skin around a source of itching. Many of the recorded findings have been inconclusive or conflicting. Aspects of particular interest that have been undecided include the relationship of the three phenomena to one another and the parts played in their production by the neural mechanisms of pain and touch. Knowledge on these and other aspects of the subject appears to have been advanced appreciably by the recently reported work of three experienced investigators in this field, David T. Graham, Helen Goodell and Harold G. Wolff.[1] Their investigations indicate that all three phenomena are mediated by the same neural structures and that, contrary to a hitherto widely held view, these structures are exclusively those which transmit pain. Touch receptors and fibres appear not to be involved. A component of tickle and of "itchy skin" does appear to be associated with touch, but this component is regarded by Graham, Goodell and Wolff as essentially extraneous, and it is noteworthy that they found both phenomena occurring in the absence of touch sensation. It is now fairly generally agreed that pain elicited in the skin has two main components—the first short and sharp, the second more prolonged and severe, to use the description given by Best and Taylor in their book "The Physiological Basis of Medical Practice". These sensations are also described respectively as "pricking" and "burning". According to the observations of Lewis and Pochin (recounted by Best and Taylor) on this phenomenon, pain impulses are conveyed from the skin by two sets of nerve fibres, one of which is rapidly conducting, the other with a much slower conduction rate; these respectively appear to mediate the two pain sensations. Graham, Goodell and Wolff have demonstrated two subjectively distinguishable components in the sensation of itch, one pricking and the other burning, and they have concluded that these correspond to the two kinds of cutaneous pain and are mediated respectively by the two types of nerve fibres involved in the transmission of pain from the skin. Moreover, they are satisfied that cutaneous tickle and the sensation elicited from "itchy skin" do not differ qualitatively from itch, except by the addition of an awareness of movement and are mediated by the same neural structures.

A number of interesting observations were made by Graham, Goodell and Wolff on the relationship between itch, "itchy skin" and tickle on the one hand and associated cutaneous pain on the other. It was found that when itching was present the pain threshold of the site was lower than it was in the same area of skin during itch-free intervals. As is pointed out, these observations are consistent with the view that stimuli which give rise to itching activate pain endings in the skin at a stimulation intensity below the pain threshold. Another finding, common to itch, "itchy skin" and tickle, was that the phenomenon was abolished by pin pricks in the adjacent area of skin. In the case of itch, this abolition is possible if the skin is pricked anywhere in the dermatome which contains the site of itching. This bears on the universally known fact that scratching relieves itch. It has been held that the relief so obtained results from replacement of the itch with frank pain; but in these experiments the effective painful stimulus was brief and far removed, and the itch did not return until some time after the pain had subsided—all findings, Graham, Goodell and Wolff claim, most readily explicable mainly in terms of central rather than peripheral processes. Similarly explicable is the observation that itching (as well as "itchy skin" and tickle) does not occur in areas of secondary hyperalgesia produced by painful stimulation of a cutaneous nerve with faradic current. The thesis put forward in this paper to explain all the findings is that the sensation of itch results from the presence in the spinal cord of impulses travelling in circuits of internuncial neurons, with a consequent patterned discharge up the spinothalamic tracts. Such circuits, it is suggested, are established when peripheral pain nerves discharge into the spinal cord at a low frequency—the result of weak stimulation. The presence of the resulting pattern of discharge in the brain is postulated as a necessary condition for the perception of itching. The corollary to this is that when the circuits are broken up—for example, by strong stimulation in the same dermatome (pin prick, scratching)—the orderly pattern is temporarily destroyed and itching ceases. It would presumably require an appreciable time for it to be reestablished, a time represented by the interval after pin-pricking before itching is again perceived. All the evidence produced goes to support the view that tickle and "itchy skin" (which though differently produced are subjectively indistinguishable and otherwise alike) depend on much the same neural mechanism as itch. Some of the experimental findings recorded do not accord with those of previous investigators, and perhaps they will be challenged. For example, Graham, Goodell and Wolff make no comment on the claim of Ehrenwald and Königstein (which they quote) that they had found two cases of syringomyelia with loss of pain but preservation of touch and itch, and one case in which pain was intact but touch and itch were absent. However, Graham, Goodell and Wolff's conclusions are based on the results of their own experiments, which they describe and of which in many cases they were the subjects, and they seem to have made out a good case. They have certainly made a stimulating contribution to an obscure subject.

[1] *The Journal of Clinical Investigation*, January, 1951.

## AS IT WAS, SO WILL IT ALWAYS BE.

MEMBERS of the medical profession are in most respects no different from other people in the community. Like other folk, they want to have a voice in the making of decisions which concern them. Sometimes they demand plebiscites; sometimes they want special meetings to be called. Members of Branch Councils know only too well what kind of a response attends these measures. If a circular is sent to members asking for replies to certain questions, a response from 40% is the usual expectation, and there is no disappointment. When the Medical Directory of Australia for 1948 was being compiled cards were sent to medical practitioners for completion; in some instances more than one card was sent and no reply was received. Many said that they had not received cards. Some practitioners who did send replies failed to realize that they had done so. Executive officers who are faced with this kind of situation "groan within themselves" and know that whatever they may do, not much change will take place. It has always been thus and always will be.

This was shown recently when a volume printed in 1771 was discovered and brought to the office of this journal. The book was a "Life of Christ", by the Reverend John Fleetwood, D.D. It was printed for J. Cooke at Shakespear's Head, Pater-Noster-Row, London. At the back was a list of subscribers which carried the following footnote:

> The publifher of this work hopes that no Subfcriber will take it amifs, if he finds his name omitted in this lift, as he has not been able to procure near one half of them, notwithftanding he gave a printed notice for that purpofe, and delayed the publication feveral weeks entirely on that account. It is alfo hoped that no gentleman will be angry if he finds his name fpelt wrong, as they are all printed exactly as they were delivered by the hawkers and others.

Branch officers who hear demands for plebiscites, and even those who make such requests, might remember what J. Cooke had to put up with in 1771.

# Abstracts from Medical Literature.

## MEDICINE.

### Fingerprints and Mercury Poisoning.

T. H. BLENCH AND H. BRINDLE (*The Lancet*, February 17, 1951) state that two police officers heavily engaged for years in fingerprint detection with mercurial powder were examined, but no clinical signs of mercurial poisoning were detected by several independent observers. The average daily urinary excretion of mercury determined over a period of two months (excluding holiday periods) when no precautions were taken was 425 microgrammes and 152 microgrammes respectively. By care in the washing of the hands and the wearing of overalls these amounts were reduced by three-quarters. The authors suggest that before the use of mercurial powder for fingerprint detection is considered as representing a definite risk, fairly protracted observation should be made on each of a large number of people who are prepared to exercise care in taking simple hygienic measures. There is some evidence that the urinary excretion of mercury can be used to supply information about the amount ingested.

### Hollow Chest as a Cause of Cardiac Insufficiency.

R. A. DORNER *et alii* (*The Journal of Thoracic Surgery*, September, 1950) report the investigation by means of angiocardiography and otherwise of a patient with a hollow chest who had symptoms of cardiac insufficiency. It was concluded that the heart was embarrassed in its action by the depression of the sternum, and elevation of the sternum surgically brought about dramatic relief.

### Primary or Idiopathic Pleurisy with Effusion.

W. A. OILLE (*The Canadian Medical Association Journal*, August, 1950) presents clinical and laboratory observations on 78 soldiers with primary or idiopathic pleurisy with effusion. He states that there was no previous history suggesting tuberculosis in any case, and the results of all chest X-ray examinations made shortly before the onset of pleurisy were negative. It was noted that the more pleural fluid cultures that were prepared, the higher was the proportion of cases proved to be tuberculous in ætiology. The character of the symptoms, clinical findings and course of the cases was essentially the same whether the results of pleural fluid culture were positive or negative for tubercle bacilli. The incidence of subsequent tuberculosis was the same in both "positive" and "negative" groups, but the disease manifested itself after a shorter interval in the "positive" group and was of a more serious and extensive nature. These differences were regarded as being due to variation of the virulence of the infecting organism, massiveness of infecting dose, and the degree of the individual's resistance. The author states that there appears to be little doubt that primary or idiopathic pleurisy with effusion is an early or primary manifestation of tuberculous infection, and the incidence of other later manifestations of tuberculosis is high. Patients with this diagnosis, whether the results of pleural fluid culture are positive or negative, should be treated as having tuberculosis, with a prolonged period of supervised bed rest, probably three or four months, after clinical signs of active infection have subsided.

### Peptic Ulcer.

D. J. SANDWEISS *et alii* (*The Journal of the American Medical Association*, December 23, 1950) discuss hormone studies in peptic ulcer. They state that two patients with duodenal ulcer were treated with cortisone, and two with pituitary adrenocorticotropic hormone; 100 milligrammes of cortisone were given twice daily for two days and then once daily. One patient was relieved of symptoms for nine months, the other was not improved. In the other two cases 100 milligrammes of pituitary adrenocorticotropic hormone were injected intramuscularly daily. One patient became worse, the other patient became symptom-free, but symptoms recurred a few days after the patient's discharge from hospital.

### Scleroderma.

J. P. TURNER AND F. R. SCHMIDT (*The Journal of the American Medical Association*, December 30, 1950) describe the treatment of scleroderma with intravenous injection of 0·1% and 0·2% solutions of procaine hydrochloride in 10% dextrose in water; 0·5 to 0·8 gramme of procaine was injected daily for some days and then twice weekly with great improvement. In one case 4·4 grammes were given in seventy days, followed by biweekly infusions of 0·6 gramme for five weeks. In another case up to 12 grammes were given in fifty-seven days. These patients all improved greatly in mobility and colour of skin. Ulcers present over bony prominences were not affected. One patient showed sensitivity to the drug, in the shape of high fever, and could not be desensitized.

### Cerebral Tumours.

L. DAVIS *et alii* (*The Journal of the American Medical Association*, December 23, 1950) discuss the use of radioactive diiodofluorescein in the diagnosis and localization of central nervous system tumours. Stable radioactive diiodofluorescein (1·1 millicuries of an 8% to 10% solution) was injected intravenously, and the uptake in the brain was determined by a single-channel differential radiation localizer, which comprises a Geiger-Müller tube, a lead shield, a radio-counting rate meter, and a mechanical graphic recorder. The radioactivity of the surface of the head is recorded for two hours. The counting rates are from 200 to 1200 counts per minute. A normal range of concentrations has been determined for each position on the surface of the head, of which 32 sites are counted. If the concentration at all sites is greater than the upper range of normal by 100 to 600 counts per minute, a tumour of the brain should be suspected. For localization, comparisons are made with the symmetrical position on the opposite side of the skull and adjacent positions on the same side. In the series of patients studied (200), there were 95 histologically verified space-occupying lesions of the central nervous system, 40 gliomata, 13 meningiomata, 13 carcinomata and sarcomata, six tumours of the spinal cord, seven tumours of the hypophysis, two hæmangioblastomata of the cerebellum, one acoustic neurinoma, one melanoblastoma, four subdural hæmatomata, two cerebellar abscesses, and other lesions. Of these 91% were diagnosed accurately, and only four diagnoses were completely inaccurate. The authors state that this method gives 95% accuracy.

### Renal Phosphatic Calculi.

E. SHORR AND A. C. CARTER (*The Journal of the American Medical Association*, December 30, 1950) describe the use of aluminium gels for renal phosphatic calculi. They state that these calculi have a tendency to recur, before or after operation. As a rule acid-ash diets or acidifying agents are given for prophylactic reasons. Urinary tract infections, difficult to eliminate, often accompany phosphatic stones. These infections prevent success in prophylaxis. Aluminium hydroxide gels cause changes in the chemical reactions of the urine, unfavourable to precipitation of phosphate ions, by forming insoluble aluminium salts in the intestinal tract, with corresponding diminution in phosphates available for excretion in the urine. With impaired renal function (hyperphosphatæmia and hypocalcæmia) these electrolytic aberrations are often corrected, with improvement in renal function, by the use of aluminium gels. The authors gave "Amphojel" by mouth to 22 patients with phosphatic stone in doses of 90 to 200 millilitres, or 40 "Amphojel" tablets daily, or 80 to 180 millilitres of "Basaljel" daily. Constipation was controlled by liquid paraffin, cascara or milk of magnesia. "Basaljel", aluminium carbonate gel, was one-third more effective by volume than aluminium hydroxide gel in reducing urinary phosphorus. A dose of 25 millilitres of "Amphojel" is said to be equivalent to three (0·6 gramme) "Amphojel" tablets. However, an amount of one and a half times this number was necessary to give results equal to those achieved with "Amphojel". The urinary phosphorus output falls under this aluminium regime from 700 milligrammes to 300 or 400 milligrammes per twenty-four hours. Treatment with aluminium was continued for as long as seven years without ill effect. Generally the results of this treatment were satisfactory in preventing recurrences of symptoms in the 22 patients in this series.

### Clinical Disorders of the Neurohypophysis.

T. FINDLEY (*Annals of Internal Medicine*, December, 1950) discusses clinical disorders of the neurohypophysis. It is stated that *diabetes insipidus* is due to a defect in the supraoptico-hypophyseal tract, which is the functional antidiuretic unit. The adrenal cortex seems to be antagonistic to the hypophysis. Hypothalamic disorders also cause obesity by increasing consumption of food. Hyperglycæmia results from pancreatic disease, and lesions of the neurohypophysis may have the same effect. *Diabetes insipidus*

is associated with destruction of the supraoptic nuclei and atrophy of the neurohypophysis. The adrenal cortex counterbalances the effect of the neurohypophysis, with regard to excretion of water and salt. Gonadal failure is related by the author to lesions of the hypothalamus or the adrenal cortex, and hypertension to lesions of the adrenal cortex or the hypophysis. He concludes that pituitrin is anti-adrenocortical, and that obesity, *diabetes mellitus* and *diabetes insipidus*, gonadal failure and hypertension may be either hypothalamic or adrenocortical in origin.

### Amœbiasis.

C. F. GUTCH (*Annals of Internal Medicine*, December, 1950) describes the treatment of amœbiasis with aureomycin. He states that 0·75 to 1·0 gramme of aureomycin was given orally every six hours up to a total of 28 grammes to proved sufferers from amœbiasis. Symptoms were relieved quickly. The results of stool examinations were negative at the end of treatment. Two patients had heavy infestations of trophozoites after six weeks' spell. These were given a second course of aureomycin and the results of stool examinations became negative. No recurrences were noted within five months.

### Hepatitis Transmitted by Tattooing.

B. F. SMITH (*The Journal of the American Medical Association*, November 25, 1950) found that 18 out of 26 men suffering from hepatitis in a military hospital had been recently tattooed. Seventeen of the men had been tattooed at one place, and it was found that a virus could have been propagated from there through the bottles of dye or through a bottle of spirit used for wiping the skin.

### The Role of Coronary Arterio-venous Communications in Cardiac Infarction.

S. HIRSCH (*Acta medica Scandinavica*, November 10, 1950) describes coronary arterio-venous communications, detected histologically, in the heart of man. The presence of neighbouring nerve fibres suggests to the author that the communications may be subject to nervous influence. He also adduces experimental evidence suggesting that the small branches of the coronary vessels play a particular role in the production of myocardial infarction by emotional shock in rats.

### Postural Hypotension.

D. VEREL (*British Heart Journal*, January, 1951), from analysis of observations of three patients subject to postural hypotension, adduces strong evidence that the disorder is due to a central failure of the carotid reflex.

### Worm Infestations Treated with Cashew Nutshell Oil.

F. W. EICHBAUM *et alii* (*The American Journal of Digestive Diseases*, November, 1950) have found that the oil of the shell of the cashew nut (*Anacardium occidentale*) has a pronounced vermicidal effect in human hookworm and whipworm infestation and causes only mild purgation and no intoxication. They administered the oil in the morning to patients with empty stomach in doses of four to six grammes in gelatine capsules. Hookworm infestation was cured in 14 out of 22 cases—in five refractory cases the patients belonged to the same family, and domestic reinfestation was a possibility. In three cases of whipworm infestation the oil effected a cure. The vermicidal effect of the oil was potentiated by its combination with hexylresorcinol.

### Anthrax Treated with the Newer Antibiotics.

H. GOLD AND W. P. BOGER (*The New England Journal of Medicine*, March 15, 1951) describe eight cases of cutaneous anthrax, all successfully treated with the newer antibiotics, two with aureomycin, four with chloramphenicol and two with terramycin.

### The Electrocardiogram in Juvenile Rheumatism.

L. M. TARAN AND N. SZELAGYI (*British Heart Journal*, January, 1951) show that in rheumatic children a close correlation exists between the duration of the electrical systole (*QT* interval) and the extent of cardiac disability resulting from acute rheumatic carditis. The longer the time that the *QT* interval remains abnormally prolonged, the more extensive the total cardiac damage. The upper limit of the normal duration of the heart's electrical systole is taken to be 0·404 second.

### Atrial Flutter.

G. R. HERRMANN AND M. R. HEJTMANCIK (*American Heart Journal*, February, 1951), who summarize the results of their treatment of 83 episodes of atrial flutter, think that digitalis is the drug of choice in the management of this disorder in patients with serious organic heart disease or cardiac decompensation or where there is a specific contraindication to quinidine. It usually acts by instituting atrial fibrillation, which reverts to sinus rhythm on withdrawal of the drug, especially if quinidine is then given. Apart from their greater rapidity of action, the pure glucosides of digitalis were not as a rule any better than the leaf. Quinidine sulphate alone restored sinus rhythm in selected patients without cardiac decompensation or grave organic heart disease, particularly if the atrial flutter was of short duration. Prognosis is poor when atrial flutter compensates myocardial infarction.

### General Anæsthesia and the Heart.

M. JOHNSTONE (*British Heart Journal*, January, 1951) has made electrocardiographic studies in 60 healthy subjects during anæsthesia. It has been demonstrated that cardiac inhibition to the point of complete cardiac arrest may occur as the result of inhaling cyclopropane or ether. The degree of inhibition varies directly with the irritancy of the inhaled vapour and appears to be due to the stimulation of vagal nerve endings in the air passages. Atropine will prevent the more serious degrees of inhibition in all except the most vagotonic subjects. The simultaneous intravenous administration of atropine and "Neostigmin" to anæsthetized patients is dangerous since it is liable to lead to ventricular tachycardia. The muscular relaxation obtained with the initial dose of curare should be maintained by anæsthetic agents and not by the administration of more curare in so far as abdominal surgery is concerned. This procedure eliminates the need for "Prostigmin".

### Streptomycin Therapy in Tuberculous Empyema.

O. CHRISTENSEN (*Acta tuberculosea Scandinavica, Supplementum* XXVI, 1950) discusses the treatment of tuberculous empyema by means of repeated intrapleural injections of streptomycin. He quotes good results from this treatment and recommends that the streptomycin should be applied locally in such a large amount of saline that the pleural surface is in continuous contact with the remedy to the greatest extent possible. Discussing the rate of absorption of streptomycin from the pleural cavity, he states that when a graph is constructed with the logarithm of the concentration of streptomycin in the fluid as ordinates and with the time after injection as abscissæ a straight line is produced; thus two samples taken on successive days allow of an estimation of the concentration on subsequent days.

### Aureomycin for the Common Cold.

R. J. HOAGLAND *et alii* (*The New England Journal of Medicine*, November 16, 1950) state that there was no significant difference in the proportion of cures reported by patients with colds who received aureomycin and by those who received a placebo. About the same proportion of patients treated with aureomycin and placebo reported slight or no benefit. About half the patients receiving the inert material reported either moderate improvement or a cure within twenty-four hours.

### The Rice-Fruit Diet for High Blood Pressure.

C. B. CHAPMAN *et alii* (*The New England Journal of Medicine*, December 7, 1950) state that there is reason to believe that when the rice-fruit diet is continued for long periods a considerable loss of body protein occurs. The addition of salt-free protein to the diet causes prompt cessation of weight loss, but in no way interferes with the depressor effect of the diet. The addition of salt, on the other hand, causes prompt return of symptoms, and in hypertensive subjects elevation of the blood pressure. A rapid decline in the level of the serum cholesterol occurs in patients receiving the rice-fruit diet. The authors believe the use of the unmodified diet over long periods to be objectionable on theoretical grounds and possibly dangerous.

### Hypothyreoidism due to the Administration of Thyreoid.

M. A. GREER (*The New England Journal of Medicine*, March 15, 1951) has investigated by the use of radioactive iodine the effect upon the function of the thyreoid gland of administering thyreoid by mouth. Pronounced depression of the gland's function could be produced in one week, usually with a dose not exceeding three grains daily. The function returned to normal within two weeks of discontinuing the drug as a rule, although occasionally it took longer. It returned to normal as rapidly in those who had been taking thyreoid for years as in those who had received it for only a few days.

# Medical Societies.

## SYDNEY INSTITUTE FOR PSYCHO-ANALYSIS.

THE provisional opening meeting of the Sydney Institute for Psycho-Analysis was held on May 3, 1951, in the Stawell Hall of The Royal Australasian College of Physicians, 145 Macquarie Street, Sydney, DR. T. M. GREENAWAY in the chair.

Dr. Greenaway opened his chairman's address by quoting the words of Thomas Hardy: "More life may trickle out of man through thought—than through a gaping wound." He went on to say that in the formation of a Sydney Institute for Psycho-Analysis was seen the first serious attempt to link the study of the subject in New South Wales with similar activities in Melbourne and abroad. The choice of a general physician as chairman for the meeting indicated, he believed, the hope that the work would receive recognition by the general body of the profession—a recognition not readily given, in fact strangely withheld, by many leaders in that profession whose general education one would have thought would promote—if not cooperation—at least a more tolerant attitude. The interest taken by doctors in the work following the researches of Freud varied from mild acceptance by many psychiatrists, and by those interested in psychosomatic medicine, to frank and unrelenting hostility by quite a large number. For some of those attitudes Dr. Greenaway thought that psychiatrists and analysts were themselves to blame. The aloofness of many psychiatrists in the past had led many practitioners to regard them as merely more charitably minded doctors, who seldom became short-tempered with their patients and, possessed of infinite patience, carried on for infinitely long periods with their patients under bromides, confidently waiting for them to get better—and always ready with a complete explanation when they did not! The analysts themselves were a still more exclusive body. Every practitioner of psycho-analysis, as, of course, they all knew, must obey the old advice of Cervantes in "Don Quixote": "Make it thy business to know thyself, which is the most difficult lesson in the world." In other words, he must himself submit to an exhaustive analysis, which, Dr. Greenaway had been told, took up to three years. The taunt of one of Dr. Greenaway's friends was that the average psychoanalyst took so much time knowing himself that he had no more left for other people. It was necessary to add that the same Cervantes in the next chapter talked about the pot calling the kettle black. It was no wonder the great Sir William Osler advised his students to read "Don Quixote".

Dr. Greenaway said that recently the general body of physicians—or many of them—had thought that they had discovered something new. They started to write about psychosomatic medicine. They had never heard of Plato or of his statement that the wise physician treated his patient, not the disease, and ministered to both body and soul. The stories of the Old and New Testaments meant nothing to them, nor had they even bothered to consider the implications or even a superficial study of comparative religion. Some of them from school days might have remembered the following passage from Shakespeare's "Macbeth:

*Macbeth*: How does your patient, doctor?

*Doctor*: Not so sick, my lord,
As she is troubled with thick-coming fancies,
That keep her from her rest.

*Macbeth*: Cure her of that:
Canst thou not minister to a mind diseas'd;
Pluck from the memory a rooted sorrow;
Raze out the written troubles of the brain;
And with some sweet oblivious antidote,
Cleanse the stuffed bosom of that perilous stuff
Which weighs upon the heart?

*Doctor*: Therein the patient
Must minister to himself.

What an attitude of resignation and defeat was implied in the last lines and how really different was the position today for those who would but see. That great humanist, Shakespeare, had known the implications of those words which he put into the mouth of the murderer Macbeth.

The truth was that a study of psycho-analysis by a group brought together many workers in the field—in much the same way as real clinical medicine brought together in addition to clinicians, chemists, physicists and a great variety of specialists, not necessarily medical graduates—which was all the better for medicine. But, Dr. Greenaway said, he would add a word of warning: psychoanalysis was not for the dilettante. He groaned when he thought of his occasional introspective patient who had "done psychology at the university". Then he was glad that he had his psychiatric colleagues not so far away! In that connexion they should think of the words of Alexander Pope in his essay on criticism:

> A little learning is a dangerous thing.
> Drink deep, or taste not the Pierian spring.
> There shallow draughts intoxicate the brain,
> And drinking largely, sobers us again.

And so, as with any other special branch requiring much study, most must look to the specialists for guidance.

Yet, Dr. Greenaway thought it could be said that many of the principles laid down by Freud and other workers had a far wider application than in the work and studies of doctors and psychiatrists. Some knowledge along those lines seemed essential for the proper understanding of subjects as far apart as the plays of the ancient Greeks or the behaviour of Mussolini or Hitler—or even the behaviour of one's own naughty children. The study of various religious movements and their ritual often gave point to modern psycho-analytical theories. He could refer only in passing to the fields of sociology and anthropology. From that point of view might be approached, in part at least, some of the greatest literary masterpieces, to say nothing of the work of artists and great composers. To that extent, then, it was not an exaggeration to say that the degree of interest taken in that field by any community was to some extent an index of its cultural development.

Dr. Greenaway then invited DR. ROY C. WINN, the prospective chairman of the institute, to read congratulatory messages from several long-established psycho-analytical institutes abroad and from well-known psycho-analysts such as Dr. Ernest Jones, formerly President of the International Psycho-Analytical Association, and Anna Freud.

DR. FRANK GRAHAM congratulated all associated with the Sydney Institute on behalf of the Melbourne Institute for Psycho-Analysis, which, he said, had been founded ten years before, largely through the generosity of Miss Lorna Traill. He added that the Sydney Institute also was fortunate in receiving financial aid from an anonymous donor, and in having the cooperation of psychiatrists just as they had had in Melbourne.

After general remarks about psycho-analysis as a science, Dr. Winn outlined the aims of the Sydney Institute, which, like the Melbourne one, was closely associated with the British Psycho-Analytical Society and with its subsidiary, the London Institute of Psycho-Analysis, which was recognized by the parent body of the British Medical Association as the psycho-analytical training centre for medical graduates in Great Britain. He added that the course took four years.

Dr. Winn explained that the nucleus of a psychoanalytical institute was the training analyst (or training analysts), and that the Melbourne Institute was centred on Clara Geroe, whom he welcomed on behalf of the Sydney Institute. The Sydney Institute, he went on to say, depended on another training analyst, Andrew Peto, recently arrived in Australia.

In addition to the training of candidates, Dr. Winn said, the institute intended to conduct courses of study for psychiatrists and other medical graduates, to foster research, to found a library, to start a child guidance clinic, to study group therapy, and to pursue the traditional psychoanalytical interest in anthropology, education, sociology, and psychometrics; in short, to make psycho-analysis more readily available to the general community.

Dr. Winn said that in addition to Andrew Peto and himself, Dr. S. Fink would be a local director of the institute, and that interstate directors would be Clara Geroe (M.D., Budapest) and Dr. Frank Graham, of Melbourne, together with Dr. Harry Southwood, of Adelaide, who also was present. Dr. Winn added that a number of Sydney psychiatrists would become honorary councillors of the institute; they were Dr. D. W. H. Arnott, Dr. A. T. Edwards, Dr. G. L. Ewan, Dr. W. H. Fraser, Dr. E. T. Hilliard, Dr. G. A. Lawrance, Dr. J. A. McGeorge, Dr. J. N. Main, Dr. E. Sydney Morris, Dr. H. M. North, Dr. Irene Sebire, Dr. C. H. Swanton and Dr. G. B. R. Wooster, most of whom were at the meeting.

Dr. Greenaway then introduced ANDREW PETO, who, he said, had graduated medically in Budapest, had until recently been secretary of the Hungarian Psycho-Analytical Society,

and for personal reasons had preferred to come to Sydney rather than become a training analyst in the London Institute of Psycho-Analysis.

Andrew Peto, speaking on the subject of war neurosis, said that observations of war neurosis in World War II compared with those in World War I suggested that the syndrome had changed its main features. While thirty years previously the motor symptoms had been in the foreground, nowadays the more common symptoms were depression, combat fatigue, working disability and radical changes of the character. Those observations could be obtained particularly from subacute and chronic cases. Andrew Peto said that the paper which he was about to read attempted to elucidate the problem from a special viewpoint.

Excessive and uncontrollable movements were the most striking symptoms of "shell shock", and they were undoubtedly in close ætiological relation to anxiety which had arisen in consequence of an overwhelming traumatic experience. As a matter of fact, muscular action—as the reaction to anxiety in man—was generally the primary reaction in child and adult to traumata of all kinds. That primary reaction might be expressed by aggression—attacking the anxiety-provoking object—or by an attempt to escape from it. Incidentally in the light of his clinical experiences, the speaker considered being petrified with anxiety a secondary defence measure. He went on to say that taking muscular action into consideration, they might be astonished by the fact that the neuroses had been showing a decrease of motor symptoms in the course of the centuries. The prophets of antiquity, witches and other possessed individuals, during the Middle Ages had had in common uncontrollable agitation, it being their most characteristic symptom during ecstasy. The medicine man and the werewolf, as well as those participating in Saint Vitus's dancing and sectarians of all religions, betrayed their relation to God or demon through agitated ecstasy. The somnambulism of the moonstruck—at one time a fashionable disease—was also dominated by a special activity (walking at night). The hysteria of the last century had shown abnormal activity or—as reversal—functional paralysis; the speaker referred to the *"grand hystérie"* of Charcot. In remarkable contrast to the past, the present practice abounded in neurotic character formations, motor symptoms having largely disappeared.

The speaker thought that his cursory outline had already sufficiently indicated the probability of the statement that disabilities of identical ætiology had been changing their symptomatology through the ages, and that they were entitled to suppose the existence of "fashionable" syndromes which prevailed at given stages of civilization.

The next question to be answered related to what was the meaning of the motor symptom in neurotics in general and what were the dynamic sources from which it originated. The problem might be approached by an analyst from two angles, namely: (i) psycho-analytical experience with adults and children; (ii) the explanation of the motor activity given by the possessed and by the people who surrounded them.

Both education and analysis offered many opportunities to observe children's extreme liability to fear provoked by the gestures of adults, which were linked with the parents' commands and emotional reactions. That fact was due to the great biological and sociological gap between adult and child, whose psychological difficulties had been brilliantly discussed by Ferenczi in his paper on the confusion of tongues between the adult and the child. Even gentle and apparently unobtrusive gestures—if unexpected—might arouse fear in the child. The great physical and mental difference between adult and child, which the latter had to bridge over in long years of development and hard social training, handicapped the child in adequate understanding of the adults' attitude towards one another and towards him. A further and even more important source of misunderstanding was the phantasy life and the magic thinking of the child, which implied—on his part—a different evaluation of his experiences at different stages of his growth. Attention should be directed to the widely spread and rather common series of traumata due in Freud's opinion to the primal scene, namely, the observation of parental intercourse. The misinterpretation of that scene and the resulting anxiety were due to two factors. The child could not understand it and therefore was frightened. The primal scene was interpreted by him—as a rule—as destructive aggression on the part of the father.

Leaving the field of clinical observation, the speaker said that he would like to draw his listeners' attention to the second point, that of the cultural phenomenon of "being possessed by the supernatural". Whether he or she was possessed by God, like the prophets of the Bible, by the devil like the witches, by any kind of demon like the medicine-man, or by some special emanation from God like many sectarians of the present age—the possessed, as well as his group, was convinced that the ecstasy was the outward sign of the uncanny supernatural in the human being. That ecstasy was very often induced by exaggerated and rhythmical movements, which were at the commencement voluntary, but became out of control in their course. The ecstasy represented a state in which the supernatural was thought to take possession of the human being and to act through his body and soul. The motor activity was the supernatural being's activity and so the cause of good and bad achieved by the possessed, who became—just for that reason—honoured or hated. Though the motor activity was but one quality of the possessed, it was the most conspicuous expression of the supernatural's aggression. The psychological process was identification with the father or mother imago, and especially with their aggression; the son introjected, took into his ego and superego, the aggressive father or mother.

The speaker said that he would like to give some explanation of those terms, as it was of importance for further understanding.

The concept "ego" covered the function of the psyche that had a double role. One was to handle reality and to maintain the optimal relation to it. The other was to face the onslaught of conscious and unconscious drives, to master them in order that those that were acceptable should be satisfied and those that were irreconcilable should be checked. That difficult function of the ego was partly conscious, but its most important activities were unconscious.

The superego was a specific differentiation of the ego—the unconscious representative of parental authority. It was the incorporated function of all the demands and commands that had been imposed upon the child in the course of his development. It was the unconscious part of the function which was known and felt by all as the "conscience".

Two peculiarities of the superego were of the greatest importance. Firstly, it was not a copy of the parents but their picture, their imago, as the child felt it; so it might be very different from the personality of the parents. Secondly, every child experienced the parental restrictions as aggression directed against him. All the commands and demands were felt unconsciously as threats and as harm done to him personally. This aggression of authority from outside was preserved in the superego, which exerted its ruling of the ego from inside. Clinical observation of child and adult proved that the unconscious aggression and power of the superego were decisive factors in the development of mental disorders. The superego helped the ego in its struggle against the impact of the instincts; at the same time it threatened and punished the ego whenever it gave way to instinctual desires that were in contradiction to the superimposed restrictions. It was clear that the harmonious or disharmonious relation of ego, superego and instincts determined the happiness or unhappiness of every individual as far as the inner mental structure and function were taken into consideration.

The means by which the ego developed and coped with both reality and instincts were manifold. Two of the most important ways were introjection and projection. The speaker had hinted at their functioning when describing the development of the superego. The love or hate objects of the outer world had to be faced and coped with. One way was to take some quality of them into one's own personality. For instance, a child might make his own the gestures or modulation of voice of his parent. A much more complicated process was—as previously described—the establishment of the superego, as a way to cope with the commands of the outer world. That was introjection. Another way to handle object relations was projection, which meant the attributing of one's own emotions or qualities to others: "Not I hate you, but you hate me." Again the development of the superego was an important example. The distorted imago of the parents was not a true copy of them, but was distorted and enhanced by the emotions which the child felt towards them. The emotions were "projected" on to the picture of the parents; this then became introjected and helped to build up the superego. So it preserved—among other components—the hatred once felt against the parents and projected on to them. The result of all those complicated processes was unconscious guilt.

After this brief digression the speaker said that he would like to return to the main line of the subject. Individual

analyses of adults and children gave abundant evidence for the validity of identification with the dangerously aggressive parent imago as the cause of motor symptoms (obsessional and hysterical motor symptoms, tics *et cetera*). Those were condensations and displacements resulting from introjective and projective mechanisms which aimed at dealing with the threatening parent imago. The ego attempted to overcome anxiety by using those two archaic defence mechanisms. The speaker said that he was assuming that the considerations mentioned especially applied to the shell-shocked patient, whose ego was of the kind that was particularly sensitive to the motor factors of his traumatic experience, which broke through his ego defences and, contacting previously repressed infantile experiences, achieved the suspension of repression. Early traumata experienced by the extremely weak ego of the child were revived by actual battle incidents endured by the soldier. The uncontrollable jerks and tics were signs of an archaic kind of identification with the moving and aggressive father imago which was represented by the likewise moving and overwhelming power of shell, plane or tank. Both war neurotic patients and those possessed tried to overcome the trauma of the aggressive father imago by means of identification with him in his uncanny activity.

Having discussed the clinical aspects of the problem, the speaker said that he would like to approach it from another viewpoint, that of civilization. A process which had begun in the past century had been gaining influence on present civilization since World War I; that process was motion, speed, which had become the most characteristic peculiarity of present civilization. Transport, industrial production, several popular sports, all owed their conspicuous development to the utilization and extremely intensified increase of speed. In the present age, more than ever before, motion and extreme speed had become increasingly important. Modern warfare depended mainly upon speed, as its outstanding features were planes, tanks and motor-cars. Speed had been gradually becoming the most distinctive essential of modern man; the rushing machine was an important element of the soldier's ego, and so its motion was united with his personality.

The speaker said that he would assume that the motor factors in a traumatic event had lost a great deal of their anxiety-arousing power because they had become less alien to the ego of the average individual in the course of recent decades. They could not break through the ego defences in most cases, and being under control they did not contact traumatic complexes of infantile origin that were of predominantly motor character. Thus they were prevented from coming into force as symptoms. Their traumatic force was taken over by other components of the anxiety-provoking situation, namely, those which could not be mastered by the ego with the help of its defences and which consequently played a dominant role in the symptomatology.

The speaker then went on to review the main features in the cases of patients whom he had had the opportunity to observe. They were 62 ex-soldiers with chronic war neurosis who had the following anamnesis and symptoms. It went without saying that not all anamnestic data and symptoms were found in every case, but all patients displayed at least most of those to be mentioned. The patients had the following previous history. The traumatic event did not involve any physical injury. During the traumatic experience and immediately after it, the emotion of being lost was foremost—such as being cut off from the platoon or being buried by bombardment. The trauma was represented not by a single event, but by a series of them. The treatment afforded opportunity to discuss traumatic experiences of early childhood which either had been conscious since they happened or came into consciousness against slight repression while the patients were receiving treatment; they always implied grave aggressions mostly committed by parents or other adults, such as hitting the boy with a stock-whip until he fainted, chasing the child with a knife until he collapsed exhausted, brutal frightening at night in the form of a pretended highwayman assault. There had been traumatic separation from the home in the latency period, which was either forced directly upon the child or was carried out voluntarily in ultimate despair, separation caused by death of the parents, maltreatment by parents or foster-parents, and emotional and material privations of all kinds. Hatred of the parents or their representatives was present; this was either conscious from early childhood on, or, when appearing after interpretation only, was accepted as such without much hesitation. There was a sense of guilt to a greater or less degree owing to this emotion of hatred. The actual symptoms were depression, emotional indifference towards relatives, at the same time—alternating with the indifference—fits of anger finding outlet in beating of the relatives, and varying degrees of impotence. Finally, attention was again drawn to the conspicuous difference between the cases resulting from the recent war and those from the first World War, namely, the lack of motor symptoms. The speaker went on to say that his short survey showed that although both the traumatic events in adulthood and the infantile anxiety state—which broke through the ego defences under the heavy weight of the trauma—involved a great number of motor components, the manifest neurosis itself did not show them. This absence—at first sight—could suggest that in consequence of the previously mentioned technical development the ego of the average individual had attained greater resistance to traumatic interferences. However, the survey brought forward clear evidence against that assumption. There was no doubt that the ego's capacity to overcome some traumatic components was heightened, but one was bound to find symptoms which indicated the fact that the gain on one side was overbalanced by the loss on the other. The ego of the average individual had attained a higher ability to handle the motor qualities of the threatening father imago, and that victory was expressed through the absence of motor symptoms in the manifest neurosis. Because of that victory of the son—who was the man of the present civilization—over the father imago, a great deal of sense of guilt was mobilized; in other words, the superego's pressure on the ego became intensified, and that process manifested itself in the form of other symptoms.

Another important factor that might be responsible for the increase of guilt feeling was the way in which modern warfare was fought. In contrast with the past, the soldier seldom or never met the enemy face to face. Thus, in the absence of real object relations—there was ample opportunity to tie up his actual activities and anxieties with infantile object relations that were phantasied at that age as dangerous (father and mother imago). Consequently not the actual enemy but early parent imagines might represent the danger to the unconscious part of the ego. The revived hostility against them aroused and strengthened the old guilt feelings of early childhood.

The process might be described in the following way. The trauma aroused the infantile fear of the father, which provoked the old anger and aggression towards him. The revived infantile hatred against the dangerous father mobilized the old guilt feelings of childhood, in other words, the revenge of the superego on the ego. The traumatically revived and feared aggression of the father imago and the counter-aggression did not appear as motion because, as had been pointed out earlier, the ego in the present civilization had introjected the motion and speed qualities of the father imago and become—in that direction—stronger. However, the pressure of the superego, which was the archaic representative of the father, insisted on punishment and retaliation for this rebellion against his authority. It was the self-evident consequence of the infantile development that the "child-parent relation" created permanent and similar, though unconscious, relations between ego and superego. Quite independently of reality, the former—the ego—preserved in more or less degree the weakness of the child and the latter—the superego—the powerful figure of the parents. This intrapsychic condition lent the superego the unconscious threat and limitless power over the ego. In turn the ego was in the grip of a permanent, unconscious fear of annihilation.

The clinical manifestations of the superego's function were depression, apathy and impotence. Depression meant—in the light of psychoanalytical experiences—that the aggression of the superego was directed against the ego in the form of self-reproaches, self-degradation, loss of interest in life, and suicidal tendencies. Apathy, exhaustion and inactivity indicated that the superego had won the upper-hand in the field in which the ego had originally succeeded. In consequence of modern technical development the ego introjected and integrated the motion of the father imago, but now the superego (the archaic and revengeful father representative) forced the ego to motor restriction and prevented it from using the gains of the conquest. Disturbances of potency were consequences of guilt feelings due to the hostility directed against the father imago. The superego condemned the sexual life of the rebellious ego and annihilated his male aspirations: the guilt reduced him to a small boy.

The only weapon of the victimized and agonized ego was the secondary gain of illness; his depression, inactivity and impotence exempted him from the obligations of an adult in society. This secondary process then provoked a renewed wave of guilt.

In conclusion, the speaker pointed out that he had attempted to approach a general problem, that of the change

in the symptomatology of some non-psychotic mental disorders. He had given his consideration to a special example, that of the traumatic war neurosis. That syndrome had changed its clinical feature in so far as the present-day neurosis showed the prevalence of guilt symptoms contrasting with the past when motor symptoms were in the foreground. It was assumed on the basis of a clinical and cultural analysis that that change was due to the technical development of civilization. The paramount importance of motion and speed had influenced the development of the average individual in such a way that his personality had become enriched with those qualities, and so their traumatic effect had been diminished. Consequently the average individual had been enabled to cope in a higher degree with the motor factors of a traumatic situation. They could not break through his ego defences and did not appear among the symptoms. A more general formulation of that view would be that contemporary dominant ideas had become part of the unconscious ego structure. However, that gain of the ego won by participation in technical progress was neutralized by the increase of guilt which was regarded as the superego's reaction to the ego-expansion. Strengthening of some ego qualities implied weakening of others. The basic trends of the human mind and the basic mental conflicts—in appearance at least—had not changed their character in the course of known human history.

## Out of the Past.

*In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.*

### SURGEON WHITE TO GOVERNOR PHILLIP.[1]

[*Historical Records of Australia.*]

Sydney Cove,
Port Jackson,
July the 4th, 1788.

Sir:

Among the troops, their wives and children, as well as among the convicts who have been ill, the want of necessaries to aid the operation of medicine has been most materially and sensibly felt. My duty leads me to mention this circumstance to your Excellency in order that you may use such means for their procurement as may seem most expedient.

For your Excellency's information I have taken the liberty to insert the articles in the margin[2] which come under the head of necessaries, to which I beg leave to add blankets and sheets for the hospital, none of which are in the colony, altho' they are essential and absolutely necessary. The want of them makes that observance and attention to cleanliness (a circumstance which amongst sick persons cannot be too much inculcated or attended to) utterly impossible. With respect to necessaries, our situation here, constantly living on salt provisions without any possibility of a change, makes them more necessary than perhaps in any other quarter of the globe.

I have, &c.,
JOHN WHITE,
Surgeon.

### SURGEON WHITE TO LORD SYDNEY.[1]

[*Historical Records of New South Wales.*]

Sydney Cove, Port Jackson,
New South Wales,
July, 1788.

My Lord:

The prevalence of disease amongst the troops and convicts, who on landing were tainted with the scurvy, and the likelyhood of the continuance from the food (salt provisions) on which they are from necessity obliged to live, has made the consumption of medicines so very great that the inclosed supply will be very much wanted before any ships can possibly arrive here from England. The distress among the troops, their wives and children, as well as among the convicts, for want of necessaries to aid the operation of medicines has been great. What are included under the head of necessaries I take the liberty to enumerate which are sugar, oatmeal, barley, rice, currants, different spices, vinegar and portable soup. Indeed, our situation, not having any fresh animal food, nor being able to make a change in the diet, which has and must be salt meat, makes these things more necessary here than, perhaps, in any quarter of the globe. However, the necessity of having these things sent out by the first opportunity will no doubt be strongly recommended to your Lordship's notice by His Excellency Governor Phillip. I have mentioned them lest they should escape him through the multiplicity of matters all new settlements afford. I have to entreat your Lordship will be pleased to cause the medicines to be sent from Apothecaries Hall, where they are sure to be genuine and fresh a circumstance, considering the immense distance we are off, and the length of time which it will take before we can receive them, that cannot be too much attended to. Care in the putting them up, and having them placed in a dry place is another circumstance equally to be attended to.

I have, &c.,
JOHN WHITE,
Surgeon.

[1] By courtesy of the Mitchell Library, Sydney.

[2] Sugar, sago, barley, rice, oatmeal, currants, spices, vinegar, portable soup, tamarinds.

### SURGEON WHITE TO GOVERNOR PHILLIP.[1]

[*Historical Records of Australia.*]

General Hospital,
Sydney Cove,
12th September, 1788.

Sir:

As his Majesty's Ship Sirius is shortly to sail for the Cape of Good Hope, I am induced from a sense of Duty, as well as Motives of Humanity, again to bring to your Excellencys Recollection how much the Marines, their Wives and Children, as well as the Convicts are and have been distressed when ill for the Want of Necessaries to aid the operation of Medicine, there being none sent out for the Use of the Hospital after landing, Wine excepted: those supplied by the Navy Board for the Voyage I have issued with the Strictest economy, but they have long been expended, and the time for which they were supplied elapsed.

As Returns from Europe cannot arrive here for a considerable time, I beg leave to particularize a few necessarys which are absolutely and indispensably requisite, and probably may be procured at the Cape a list[2] of which I have taken the Liberty to enclose. When necessarys are sent from England I hope Blankets and Sheets for the Hospital will not be overlooked as none were sent out although demanded.

I have, &c.,
JOHN WHITE.

## Correspondence.

### SPECIALIZATION.

SIR: There is an article in *The Lancet* of February 24, 1951, on specialization by Dr. John W. Todd dealing with various aspects of this subject in Great Britain.

Under the heading "Selection and Training of Specialists and Consultants", it is stated:

> In recent years in Britain the typical potential specialist or consultant does some house appointments after qualification, obtains higher degrees or diplomas, becomes a registrar and, after three or more years, is appointed to hospitals with the title of consultant. This process no doubt turns out men who are very knowledgeable in their subjects and (if they are surgeons) have mastered some technique. But I believe it fails to give men the ability to see the problems of medicine in proper perspective. It has often struck me that the average surgical registrar tends to be interested in operating, to the exclusion of all else. The interest of the average medical registrar, on the other hand, is partly aroused by the very rare and obscure case. . . .

[1] By courtesy of the Mitchell Library, Sydney.

[2] This list has not yet been found.

> The problem would, I believe, be largely solved if all clinical registrars were selected from the ranks of the general practitioners. The minimum period spent in general practice should be, say, three years. . . . Every medical student who intended to become a clinician would then naturally acquire the idea that the purpose of his training was to make him a competent general practitioner.

Since the Royal Colleges have come into being and become established, is it not right to say the same thing as has been noticed in Great Britain is happening here? Do not many young graduates proceed to the practice of specialization without ever having cared for a patient outside a hospital? It would appear that there is a great deal of truth in the statement that specialists look after diseases and general practitioners look after patients.

The leading physicians, surgeons and specialists of the past suffered no disability because they learned to look after the patients as general practitioners before rising to eminence as physicians, surgeons and specialists.

Osler asked the question: "Wherewithal shall the young man prepare himself should the ambition arise in him to follow in the footsteps of such a teacher as, let us say, the late Austin Flint—the young man just starting, and who will from 1915 to 1940 stand in relation to the profession of this city and this country, as did Dr. Flint between 1861 and the time of his death?" Osler goes on to describe the various ways in which the young doctor wishing to qualify as physician shall spend his time for at least ten years.

Further on, Osler says the young graduate must put his emotions on ice. There must be "no Amaryllis in the shade" and he must beware of the "tangles of Neæra's hair". How does this apply to conditions half-way through the twentieth century? The addresses from which these quotations are taken were written at the end of the nineteenth century.

At the present time no Queensland graduate can call himself a specialist (by which term one includes physicians, surgeons, gynæcologists, ophthalmologists *et cetera*) unless he has a higher degree or diploma. Graduates taking a degree in surgery—and there have been quite a few of them in Queensland—are not so badly off, if they live in Brisbane, as they are able to take a course of study and lectures and can sit for the degree. Graduates from the country are not so well placed. The M.D. degree may only be obtained by thesis, and so far only one graduate has obtained the degree (a senior lecturer in the department of physiology). No graduate has so far obtained a diploma. There are only two subjects in which the diploma may be taken, namely, psychological medicine and ophthalmology.

The question of diplomas is a very vexed one, as no courses are given in the daytime, and, moreover, most of the lectures and demonstrations fall upon members of the university staff, who are very overworked with teaching undergraduates in the usual departmental work. Once again, the countryman is at a distinct disadvantage.

There is a school of thought that is of the opinion that graduates seeking higher degrees should go to other States or abroad in order to get the advantage of other ideas than those of their own university. Many university lecturers feel that, if a graduate student wishes to take a higher degree or diploma, he should take off the necessary time from his general practice and devote himself to study for the diploma. On the other hand, graduates feel, especially those who marry early, that such a practice is impossible for them and they are denied the chance of specializing.

The net result of all this is that owing to legislation the number of people who are able to call themselves specialists is getting relatively fewer. The condition could be improved to a certain extent by the creation of registrarships at the hospitals and making financial grants to those who are prepared to sacrifice the time for the purpose of higher study.

Dr. Todd says: "Such qualities as good judgement, the ability to see the patient as a whole, the ability to see all aspects in the right perspective, and the ability to weigh up evidence are far more important than detailed knowledge of some rare syndrome, or even the possession of an excellent memory and a profound desire for learning."

Can it be said whether the young specialist today in all cases has, by training, those attributes which are the mark of a good doctor? Is it not a fact that many of them have never personally been responsible for treating a patient outside a hospital? Without this personal responsibility they rarely attain the qualities of the physician and surgeon of former days.

The matters that have been mentioned create a very difficult problem. Would it not be a good thing if the Royal Colleges and the universities considered not only making it obligatory for all practitioners seeking higher degrees and diplomas to spend a period in general practice, but also took up the question of whether we are doing all we can to provide the specialists (using this term in the wider sense) that are necessary for the service of the community?

Yours, etc.,

E. S. MEYERS.

The University of Queensland Medical School,
Herston Road,
Brisbane, N.1.

June 13, 1951.

## SOME POINTS IN THE MANAGEMENT OF VARICOSE VEINS.

SIR: Dr. J. N. R. Stephen's short letter on this subject appearing in THE MEDICAL JOURNAL OF AUSTRALIA on June 2, 1951, is full of pith. It raises most of the problems connected with the treatment of varicose veins and I shall try to be brief in my reply.

The length of operation is forty-five to sixty minutes for each limb. This varies according to the number of incisions and the size of the patient.

Local anæsthetic (1% "Novocain" with 1:100,000 adrenaline) is used in the vast majority of cases. General anæsthetic is used if the patient is very nervous, or asks for it.

Generally the patient is in hospital seven days and another seven off work. These figures vary considerably and depend on many circumstances—that is, the extent of the operation, type of patient (thin, fat, nervous or phlegmatic) and type of work. Patients can go home in one, two or three days and resume work. One slim, calm young lady had her operation on Friday morning, spent the week-end at home, and went to work as a secretary on Monday.

One patient had a deep vein thrombosis with pulmonary complications, due to using incorrect strength of solution. Ten years ago, after the use of a soapy solution for retrograde injection, the patient had extreme redness of the limb and a temperature of 103° F. This happened only once as diluted solutions have been used ever since. Sometimes mild redness and swelling will occur when estimation of dose and strength of injection has not been quite correct, but the condition is only transitory, and not serious. Infection of the groin wound is prone to occur, and strict asepsis is necessary at operation—not "minor op" technique (Lawes, 1940). One patient's foot was noticed to be pale and cool when examined at the end of the operation. It recovered quickly with intravenous injection of "Etamon" and application of local heat.

I agree that a retrograde injection can produce all the complications mentioned. It has done so, and will continue to do so if incorrect methods are used. It is a deplorable fact that severe pain, high temperature and confinement to bed are accepted as unavoidable accompaniments of the operation. One hears it on all sides. Patients tell of friends who suffered agony; surgeons mention the fact; physicians and other non-operating practitioners fear to recommend the operation to their patients; nurses and sisters warn patients what to expect. This pain and high temperature should never occur—they are evidences of poor treatment. So, too, are recurrences. Dr. Stephen is quite right—the percentage of failures is high and the lack of interest great (Lawes, 1942, 1946). When I started this work at Poplar Hospital, London, in 1938, I learnt that all tributaries must be divided at the *fossa ovalis*, as well as the long saphenous vein itself. Yet even today one still sees the operation done well below the sapheno-femoral junction. The unfortunate part of all this is that there is doubt in many minds of the efficacy of treatment of varicose veins, and many patients suffer unnecessarily because they do not know they can be cured. I do not quite understand Dr. Stephen's last paragraph—especially the phrase "minimum of surgery". I agree that much benefit can be given to patients by skilful injection therapy, and others have thought so, too (Harvey, 1949). I am convinced, however, that no cure is possible in severe cases without such basic operative procedures as were mentioned in my article. Freedom from recurrence in three years is not evidence of cure—a longer period is required; it is evidence of relief. Good injection therapy is better than bad surgery, but good surgery is best of all, and cures the patient. Legs cease to ache and look like legs again, ulcers heal, rashes improve, bandages are dis-

carded, shopping can be done in comfort, golf handicaps are reduced—to mention a few of the advantages of such a cure.

Yours, etc.,

Sydney, June 13, 1951. C. H. WICKHAM LAWES.

### References.

Harvey, R. Simpson (1949), "Treatment of Varicose Veins", *British Medical Journal*, Volume I, page 1005.

Lawes, C. H. Wickham (1940), "An Unusual Arrangement of the Long Saphenous Vein", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 261.

——— (1942), "Varicose Veins in Peace and War", *ibidem*, Volume II, page 499.

——— (1946), "Some Problems of Varicose Veins", *ibidem*, Volume II, page 685.

# University Intelligence.

## AUSTRALIAN VICE-CHANCELLORS' COMMITTEE.

### NUFFIELD DOMINIONS TRUST: APPOINTMENTS AT OXFORD MEDICAL SCHOOL.

NOTICE has been received from the Registrar of the University of Oxford that the university has decided to invite nominations from Australia for two posts in the Oxford Medical School.

The two posts are as follows:

1. A demonstratorship tenable in any one of the following pre-clinical departments: Department of Biochemistry, Department of Human Anatomy, Department of Physiology, Department of Pharmacology. The appointee must be a graduate with experience in research.

2. An assistantship in any one of the following clinical departments: Nuffield Department of Obstetrics and Gynæcology, Nuffield Department of Orthopædic Surgery, Nuffield Department of Anæsthetics. The appointee must be a graduate and have a medical qualification.

No person shall be appointed to either post who does not intend to return to Australia for at least five years' work of a like nature. The appointment is to commence as early as possible in academic year commencing October, 1951.

The emoluments will be as follows. The stipend for a single man will be £550 *per annum*, and for a married man £850 *per annum*, both subject to United Kingdom income tax. The travel grant will be £300.

The tenure of appointment may be for two or three years, with the exception that an appointment in the Department of Biochemistry or the Nuffield Department of Orthopædic Surgery would be tenable for only two years in the first instance, and in the Department of Human Anatomy should preferably be for three years. (The University of Oxford cannot undertake to provide accommodation for the families of the persons appointed.)

Applications in duplicate, supported by the dean of the faculty of medicine in which the applicant trained or in the university with which the applicant is now associated, must reach the Secretary of the Australian Vice-Chancellors' Committee, c.o. the Australian National University, Box 4, G.P.O., Canberra, A.C.T., by not later than July 16, 1951.

Further information is available from each of the Australian universities or from the Secretary of the Australian Vice-Chancellors' Committee.

## UNIVERSITY OF MELBOURNE.

### ELECTIONS TO COUNCIL AND STANDING COMMITTEE OF CONVOCATION.

THE Registrar of the University of Melbourne has advised that the elections to the Council and the Standing Committee of Convocation of the University will take place in December, 1951. Nominations will be invited in October, 1951, and according to statute, graduates who wish to vote will need to apply for voting papers. To facilitate the applications, application cards will be distributed by the university authorities.

### DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JUNE 9, 1951.[1]

| Disease. | New South Wales. | Victoria. | Queensland. | South Australia. | Western Australia. | Tasmania. | Northern Territory.[2] | Australian Capital Territory. | Australia.[3] |
|---|---|---|---|---|---|---|---|---|---|
| Ankylostomiasis | * | .. | .. | .. | .. | .. | .. | .. | .. |
| Anthrax | * | .. | .. | .. | .. | .. | .. | .. | .. |
| Beriberi | * | * | * | * | * | * | .. | * | .. |
| Bilharziasis | * | .. | .. | .. | .. | .. | .. | .. | .. |
| Cerebro-spinal Meningitis | .. | .. | .. | .. | 2(1) | 1 | .. | 1 | 4 |
| Cholera | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Coastal Fever(*a*) | * | * | .. | * | * | * | * | * | .. |
| Dengue | * | * | * | * | * | * | .. | * | .. |
| Diarrhœa (Infantile) | .. | .. | 8(8) | .. | 2(2) | .. | .. | .. | 10 |
| Diphtheria | 17(3) | 3 | 3(2) | 1 | 8(6) | .. | .. | .. | 32 |
| Dysentery (Amœbic) | * | .. | .. | .. | .. | .. | .. | .. | .. |
| Dysentery (Bacillary) | * | .. | 4(4) | 1(1) | .. | .. | .. | .. | 5 |
| Encephalitis Lethargica | .. | 1(1) | .. | .. | .. | .. | .. | .. | 1 |
| Erysipelas | * | * | * | 2(2) | * | * | .. | .. | 2 |
| Filariasis | * | .. | .. | .. | .. | .. | .. | .. | .. |
| Helminthiasis | * | .. | * | * | * | * | * | * | .. |
| Hydatid | * | .. | * | * | .. | * | * | .. | .. |
| Influenza | * | * | * | .. | * | * | .. | * | .. |
| Lead Poisoning | * | * | .. | * | * | * | * | * | .. |
| Leprosy | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Malaria(*b*) | * | .. | .. | .. | .. | .. | .. | .. | .. |
| Measles | * | * | * | 33(3) | * | * | .. | 1 | 34 |
| Plague | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Poliomyelitis | 23(8) | 12(6) | 17(5) | 14(12) | 1 | 1(1) | .. | .. | 68 |
| Psittacosis | * | .. | * | .. | * | * | * | * | .. |
| Puerperal Fever | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Rubella(*c*) | * | * | * | * | 4 | * | .. | .. | 4 |
| Scarlet Fever | 13(6) | 34(22) | 12(4) | 8(5) | 5(3) | 7(6) | .. | 1 | 80 |
| Smallpox | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Tetanus | * | .. | 1(1) | * | 1(1) | * | * | * | 2 |
| Trachoma | * | .. | * | * | * | * | * | * | .. |
| Tuberculosis(*d*) | 17(10) | 13(6) | 9(5) | 12(7) | 5(4) | 5(1) | .. | .. | 61 |
| Typhoid Fever(*e*) | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Typhus (Endemic)(*f*) | .. | .. | 2 | .. | 1 | .. | .. | .. | 3 |
| Undulant Fever | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Weil's Disease(*g*) | * | * | 1 | * | * | * | * | * | 1 |
| Whooping Cough | * | * | * | 1 | * | * | .. | 1 | 2 |
| Yellow Fever | .. | .. | .. | .. | .. | .. | .. | .. | .. |

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from the Northern Territory.

* Not notifiable.

(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

### UNIVERSITY OF SYDNEY.

A LATE AFTERNOON RECEPTION will be held in the Great Hall of the University of Sydney on Friday, September 21, 1951, from 5.30 to 7.30 p.m. It has been arranged by the Women's Union of the University of Sydney as the Women's Union's contribution to the University Appeal. Admission is by card only, and subscription is not payable at the door. The cost of admission cards is 12s. 6d. each. Booking vouchers and admission cards are obtainable from the Secretary of the Women's Union, Manning House, University of Sydney. Telephone: MW 1758. The closing date for the securing of tickets will be August 24, 1951.

## Obituary.

### ARTHUR NORMAN HOMEWOOD.

WE regret to announce the death of Dr. Arthur Norman Homewood, which occurred on June 22, 1951, at Sydney.

### OWEN PERDRIAU.

WE regret to announce the death of Dr. Owen Perdriau, which occurred on June 20, 1951, at Wahroonga, New South Wales.

## Australian Medical Board Proceedings.

### NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1945, as duly qualified medical practitioners:

Walker, David Gerard, M.B., B.S., 1951 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Walker, John Bernard, M.B., B.S., 1951 (Univ. Sydney), Lewisham Hospital, Lewisham.

Walker, Keith Bernard, M.B., B.S., 1951 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Walsh, Desmond Michael, M.B., B.S., 1951 (Univ. Sydney), Lewisham Hospital, Lewisham.

Walsh, John Raymond Warn, M.B., B.S., 1951 (Univ. Sydney), St. George Hospital, Kogarah.

Ward, William John, M.B., B.S., 1951 (Univ. Sydney), Grafton Base Hospital, Grafton.

White, Harold John, M.B., B.S., 1951 (Univ. Sydney), Royal Newcastle Hospital, Newcastle.

Whitington, Ronald Ernest, M.B., B.S., 1951 (Univ. Sydney), Dubbo Base Hospital, Dubbo.

Wolfenden, William Horace, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.

Woodgate, Geoffrey Irving, M.B., B.S., 1951 (Univ. Sydney), Cooma District Hospital, Cooma.

Worling, Babette Josephine, M.B., B.S., 1951 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Wright, Francis Boyd, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Wurth, William Geoffrey, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.

Blank, Joseph, 4 Ormonde Street, Bondi. Registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1950.

Enis, Zenon, 63 Queen Street, Ashfield. Registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1950.

Grosslicht, Robert, 22 Cowper Street, Randwick. Registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1950.

Laszlo, John Eugene, 17 Gladswood Gardens, Double Bay. Registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1950.

Wajnryb, Abraham, 231 Queen Street, Campbelltown. Registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1950.

Hitchings, Patrick Terence Llewellyn, M.B., Ch.B., 1946 (Univ. New Zealand), Urana.

Kelly, Laurence Murray, M.R.C.S. (England), L.R.C.P. (London), 1934, M.B., B.S., 1936 (Univ. London), c.o. Bank of Australasia, Sydney.

## Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Benecke, John, M.B., B.S., 1950 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Mitchell, Robert Wentworth, M.B., B.S., 1951 (Univ. Sydney), Base Hospital, Dubbo.

Shumack, Ian Albert, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Macquarie Street, Sydney.

## Diary for the Month.

JULY 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee. Organization and Science Committee.

JULY 13.—Queensland Branch, B.M.A.: Council Meeting.

JULY 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

*New South Wales Branch* (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

*Victorian Branch* (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

*Queensland Branch* (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

*South Australian Branch* (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

*Western Australian Branch* (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

## Editorial Notices.

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